To: Beck, Nancy[Beck.Nancy@epa.gov]
From: Dourson, Michael
Sent: Mon 11/13/2017 10:13:38 AM
Subject: Re: Epi studies

Ok, let's chat about this before the briefing.

Sent from my iPad

On Nov 12, 2017, at 3:52 PM, Beck, Nancy <Beck.Nancy@epa.gov> wrote:

Ex. 5 - Deliberative Process

Also, lets make sure that the briefing that OPP staff are planning to give you is the briefing that you are interested in.

Nancy

From: Dourson, Michael
Sent: Sunday, November 12, 2017 2:59 PM
To: Beck, Nancy
Cc: Bertrand, Charlotte
Subject: RE: Epi studies

Nancy

Ok. As you well know, the decision on chlorpyrifos will likely turn on Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Cheers!

Michael

From: Beck, Nancy
Sent: Friday, November 10, 2017 2:31 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Subject: Epi studies

Mike,

For a first briefing to help get you and Charlotte up to speed, it may be helpful to

Ex. 5 - Deliberative Process

Sent from my iPhone, please excuse typos.

To: Ex. 6 - Personal Privacy (sender's personal email address)
From: Dourson, Michael
Sent: Sat 10/28/2017 12:20:35 PM
Subject: FW: Updated: Document for the Hill
2017-10-27 EPA Nominee Dr. Michael Dourson.docx

From: Bowman, Liz
Sent: Friday, October 27, 2017 9:29 PM
To: Dourson, Michael <dourson.michael@epa.gov>; Jackson, Ryan <jackson.ryan@epa.gov>; Lyons, Troy <lyons.troy@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Updated: Document for the Hill

The link was being weird for me too, so I copied the URL into the document – its here

From: Dourson, Michael
Sent: Friday, October 27, 2017 6:35 PM
To: Jackson, Ryan <jackson.ryan@epa.gov>; Lyons, Troy <lyons.troy@epa.gov>
Cc: Bowman, Liz <Bowman.Liz@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Updated: Document for the Hill

Ryan

The link I have is http://www.wcpo.com/news/local-news/i-team/i-team-mystery-of-what-was-killing-animals-sickening-children-on-nky-property-solved. However, this may be slightly different than what I send previously to Liz. On my new government computer the video at this link, which showed the letter I wrote with my signature, does not appear to be working. Unfortunately, my previous email to Liz is part of the university computer that I do not have.

Hopefully this link will work. Liz, feel free to send my prior email on.

Cheers!

Michael

From: Jackson, Ryan
Sent: Friday, October 27, 2017 6:00 PM
To: Lyons, Troy <lyons.troy@epa.gov>
Cc: Bowman, Liz <Bowman.Liz@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: Updated: Document for the Hill

The link does t seem to work on the news story. Is there another link?

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Oct 26, 2017, at 8:35 PM, Lyons, Troy <lyons.troy@epa.gov> wrote:

Ex. 5 - Deliberative Process

Also, I would capitalize the positions he's held

Looks great
Sent from my iPad

On Oct 26, 2017, at 9:23 PM, Bowman, Liz <Bowman.Liz@epa.gov> wrote:

Please review in detail and check all facts. When it's final, we need to remove the "draft" from the layout.

Thank you,

Liz Bowman

U.S. Environmental Protection Agency (EPA)

Office: 202-564-3293

<2017-10-26 Draft Myth v Reality on Dourson.docx>

To: Jackson, Ryan[jackson.ryan@epa.gov]
From: Dourson, Michael
Sent: Tue 1/2/2018 2:53:31 PM
Subject: FW: Brief meeting?

Ryan

I am sure that your schedule is packed, but do you have wee bit of time today to talk? Thanks in advance.

Mike

-----Original Message-----
From: Dourson, Michael
Sent: Tuesday, January 2, 2018 6:45 AM
To: Willis, Sharnett <Willis.Sharnett@epa.gov>
Subject: Brief meeting?

Sharnett

Does Ryan have about 15 minutes today? It can even be between 6 & 9 pm.

Thanks!

Michael

Sent from my iPhone

Cc: Beck, Nancy[Beck.Nancy@epa.gov]; Bolen, Derrick[bolen.derrick@epa.gov]
To: Hanley, Mary[Hanley.Mary@epa.gov]
From: Dourson, Michael
Sent: Wed 10/25/2017 11:21:44 AM
Subject: Re: EPA - IFRANA Meeting: October 26 at 3:30pm

Mary

Thanks!

Mike

Sent from my iPad

> On Oct 24, 2017, at 3:43 PM, Hanley, Mary <Hanley.Mary@epa.gov> wrote:
>
> Mike,
> Attached are materials for this Thursday's meeting.
> Cheers
> M
>
> From: Ringel, Aaron
> Sent: Tuesday, October 24, 2017 2:11 PM
> To: Hanley, Mary <Hanley.Mary@epa.gov>
> Cc: Beck, Nancy <Beck.Nancy@epa.gov>
> Subject: FW: EPA - IFRANA Meeting: October 26 at 3:30pm
>
> Hi Mary, see attached and below from IFRANA in regards to our meeting on Thursday.
>
> Let me know if you need any additional info.
>
> Best,
>
> -Aaron
>
> From: Amanda Nguyen [mailto:anguyen@ifrana.org]
> Sent: Tuesday, October 24, 2017 1:26 PM
> To: Rodrick, Christian <rodrick.christian@epa.gov<mailto:rodrick.christian@epa.gov>>; Ringel, Aaron <ringel.aaron@epa.gov<mailto:ringel.aaron@epa.gov>>
> Cc: Adkerson, Robert <Robert.Adkerson@mail.house.gov<mailto:Robert.Adkerson@mail.house.gov>>; Renberg, Dan <Dan.Renberg@arentfox.com<mailto:Dan.Renberg@arentfox.com>>; Neal, Aubrey <Aubrey.Neal@mail.house.gov<mailto:Aubrey.Neal@mail.house.gov>>
> Subject: Re: EPA - IFRANA Meeting: October 26 at 3:30pm
>
> Good afternoon,
>
> IFRANA is looking forward to meeting with the agency on Thursday. Attached, please find the following:
>
> • Meeting Participants
> • IFRANA Summary Sheet
> • Issues Summary
> o New Chemicals
> o LSCA Implementation
> o Relying on IFRANA as a Resource
>
> Please let me know if you have any questions or if I can provide additional information/insight.

>
> Best,
>
> Amanda
>
> Amanda K. Nguyen, J.D.
> Director, Government Affairs & Legal
> IFRANA - the fragrance industry association
> 1655 Fort Myer Drive, Suite 875
> Arlington, VA 22209
> Office: (571) 317-1506
> Mobile: (316) 461-2812
> anguyen@ifrana.org<mailto:anguyen@ifrana.org>
> This message (and any attachments) is intended only for the use of the individual or the company to which it is addressed, and may contain information that is confidential, proprietary, and privileged. If you are not the intended recipient, any use, distribution, or copying of this communication is strictly prohibited. If you received this message in error, please notify the sender and delete this message and any attachments. Thank you.
>
> From: "Rodrick, Christian" <rodrick.christian@epa.gov<mailto:rodrick.christian@epa.gov>>
> Date: Wednesday, October 11, 2017 at 2:15 PM
> To: "Renberg, Dan" <Dan.Renberg@arentfox.com<mailto:Dan.Renberg@arentfox.com>>, "Neal, Aubrey" <Aubrey.Neal@mail.house.gov<mailto:Aubrey.Neal@mail.house.gov>>, "Ringel, Aaron" <ringel.aaron@epa.gov<mailto:ringel.aaron@epa.gov>>, Amanda Nguyen <anguyen@ifrana.org<mailto:anguyen@ifrana.org>>
> Cc: "Adkerson, Robert" <Robert.Adkerson@mail.house.gov<mailto:Robert.Adkerson@mail.house.gov>>
> Subject: RE: EPA - IFRANA Meeting: October 26 at 3:30pm
>
> Thanks All,
>
> Dan and Amanda, once you have those background materials and a number of attendees, please do share that with me.
>
> Because of EPA's security policies, it will take a few minutes to get all attendees through security, signed in, and up the office. For this reason, depending on the number of attendees, I would ask that you arrive an extra 5-10 minutes early. Once you are close to EPA HQ, please give me a call so I can meet you at the East Building and we can get you into the building. For your awareness, my cell phone is (202) 578-2755.
>
> EPA's address is 1200 Pennsylvania Ave. NE, Washington, D.C. However, for your awareness, the EPA East Building is right at the corner of 12th St. NW and Constitution Ave NW. You will want to enter through the Constitution Ave Entrance. Of course, if you have any trouble finding it, always feel free to call me.
>
> Additionally, please be sure to bring your IDs to the office with you so you are able to sign in.
>
> If you have any additional questions, please feel free to call and we look forward to seeing you on the 26th.
>
> Christian Rodrick
> Special Assistant
> Congressional and Intergovernmental Affairs
> U.S. Environmental Protection Agency
> O: (202) 564-4828
>

> From: Renberg, Dan [mailto:Dan.Renberg@arentfox.com]
> Sent: Wednesday, October 11, 2017 1:08 PM
> To: Neal, Aubrey <Aubrey.Neal@mail.house.gov<mailto:Aubrey.Neal@mail.house.gov>>; Ringel, Aaron <ringel.aaron@epa.gov<mailto:ringel.aaron@epa.gov>>; Rodrick, Christian <rodrick.christian@epa.gov<mailto:rodrick.christian@epa.gov>>; Amanda Nguyen <anguyen@ifrana.org<mailto:anguyen@ifrana.org>>
> Cc: Adkerson, Robert <Robert.Adkerson@mail.house.gov<mailto:Robert.Adkerson@mail.house.gov>>
> Subject: RE: EPA - IFRANA Meeting: October 26 at 3:30pm
>
> Aaron and Christian – Thanks for your effort to facilitate this meeting, which is of great importance to the fragrance industry members of IFRANA.
>
> Aubrey and Rob – Thanks for helping get our meeting request into the right hands.
>
> We will gladly pull together some helpful background materials in the coming days so that the EPA meeting participants will have some context on the industry and some of its concerns.
>
> We are looking forward to the 26th.
>
> Regards,
>
> Dan
>
> Dan Renberg
> Partner
>
> Arent Fox LLP | Attorneys at Law
> 1717 K Street, NW
> Washington, DC 20006-5344
> 202.857.6386 DIRECT | 202.857.6395 FAX
> dan.renberg@arentfox.com<mailto:Dan.Renberg@arentfox.com> | www.arentfox.com<http://www.arentfox.com>
>
> From: Neal, Aubrey [mailto:Aubrey.Neal@mail.house.gov]
> Sent: Wednesday, October 11, 2017 1:06 PM
> To: Ringel, Aaron <ringel.aaron@epa.gov<mailto:ringel.aaron@epa.gov>>; Rodrick, Christian <rodrick.christian@epa.gov<mailto:rodrick.christian@epa.gov>>; Amanda Nguyen <anguyen@ifrana.org<mailto:anguyen@ifrana.org>>; Renberg, Dan <Dan.Renberg@arentfox.com<mailto:Dan.Renberg@arentfox.com>>
> Cc: Adkerson, Robert <Robert.Adkerson@mail.house.gov<mailto:Robert.Adkerson@mail.house.gov>>
> Subject: EPA - IFRANA Meeting: October 26 at 3:30pm
>
> Good afternoon, All –
>
> I wanted to connect everyone and start a new email chain since we have officially booked the EPA-IFRANA meeting for Thursday, October 26, 2017 at 3:30pm.
>
> By way of introduction:
>
> From the EPA, Aaron Ringel is Deputy Associate Administrator and has been working with us to coordinate the meeting with Dr. Beck and Dr. Dourson. Christian from his office will be assisting with scheduling details and logistics.
>
> From IFRA North America's Government Affairs and Legal Team, Director Amanda Nguyen and Dan Renberg will be the points of contact for attendee information and a more detailed list of discussion points for the roundtable.

>
> From Congressman Loudermilk's office, Chief of Staff Rob Adkerson and I will be attending as well.
>
> Please let me know how I can be of assistance in the coming weeks. I look forward to the roundtable!
>
>
> Best,
>
> Aubrey Neal
>
> Legislative Assistant
> Congressman Barry Loudermilk | GA-11
> 329 Cannon HOB | Washington D.C. 20515
> (202) 225-2931 | loudermilk.house.gov
>
>
> _____
>
> CONFIDENTIALITY NOTICE: This e-mail and any attachments are for the exclusive and confidential use of the intended recipient. If you received this in error, please do not read, distribute, or take action in reliance upon this message. Instead, please notify us immediately by return e-mail and promptly delete this message and its attachments from your computer system. We do not waive attorney-client or work product privilege by the transmission of this message.
> <Oct. 26 IFRANA Participants.pdf>
> <IFRANA Leave-Behind.pdf>
> <IFRANA Discussion Topics with EPA.pdf>

To: Jackson, Ryan[jackson.ryan@epa.gov]
From: Dourson, Michael
Sent: Thur 11/2/2017 12:46:38 PM
Subject: RE: RE: RE:

Yes sir!

From: Jackson, Ryan
Sent: Thursday, November 2, 2017 8:40 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: RE: RE:

9:30. Get some coffee first for yourself.

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Nov 2, 2017, at 7:52 AM, Dourson, Michael <dourson.michael@epa.gov> wrote:

ok

From: Jackson, Ryan
Sent: Thursday, November 2, 2017 6:40 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: RE:

How about 9?

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Nov 1, 2017, at 8:01 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

Ryan

Sure, my whole morning is blissfully open. Just say when...

Michael

Sent from my iPad

On Nov 1, 2017, at 7:33 PM, Jackson, Ryan <jackson.ryan@epa.gov> wrote:

Thanks for this mike.

Can we talk again tomorrow?

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Nov 1, 2017, at 1:20 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

Ryan

An attorney, Richard Bowles,* called me up to consider expert testimony in a contaminated residential site. The resident Ex. 6 - Personal Privacy was claiming

health effects and loss of real estate value due to contamination with TCE and other solvents due to a ground water plume underneath his property. I agreed and testified to the credibility of EPA's cancer slope factor. The case may not have gone to trial, since I did not have to make a court appearance. The case was No. CIV MSC 05-01725, IN THE SUPERIOR COURT OF THE STATE OF CALIFORNIA IN AND FOR THE COUNTY OF CONTRA COSTA; RON BLOCK, et al., Plaintiff; DANIEL HELIX, et al., Defendant.

Cheers!

Michael

*Bowles and Verna LLP
2121 N. California Blvd., Suite 875
P.O. Box 8180
Walnut Creek, CA 94596-8180

925 935 3300

From: Jackson, Ryan
Sent: Wednesday, November 1, 2017 1:06 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE:

I know the Johnson example. The news story laid it out really well. Can you fill me in on the facts of the TCE example?

From: Dourson, Michael
Sent: Wednesday, November 1, 2017 1:05 PM
To: Jackson, Ryan <jackson.ryan@epa.gov>
Subject: RE:
Importance: High

Ryan

Here is the contact information:

•□□□□□□□□ Ex. 6 - Personal Privacy in the story about hydrogen sulfide: Ex. 6 - Personal Privacy would likely be highly supportive.

•□□□□□□□□ Paul Dickman [redacted] attorney of record at the time of TERA's pro bono work: 859 491 7999w; Ex. 6 - Personal Privacy c. I do not believe he [redacted] attorney now, but would like be supportive.

•□□□□□□□□ Richard Bowles, the plaintiff's attorney from Bowles and Verna LLP where I testified on behalf of using EPA's TCE cancer potency value to support the plaintiff's claim of health risk: 925 935 3300. He would likely be supportive.

Cheers!

Michael

From: Jackson, Ryan
Sent: Wednesday, November 1, 2017 12:45 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE:

Can you stop by?

From: Dourson, Michael
Sent: Wednesday, November 1, 2017 12:44 PM
To: Jackson, Ryan <jackson.ryan@epa.gov>
Subject: RE:

Ryan

I am free until 2 pm today and then after about 5:30. My cell is Ex. 6 - Personal Privacy
Ex. 6 - Personal Privacy

Cheers!

Michael

From: Jackson, Ryan
Sent: Wednesday, November 1, 2017 11:26 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject:

Mike, can you give me a call? Ex. 6 - Personal Privacy

Ryan Jackson

Chief of Staff

U.S. Environmental Protection Agency

Ex. 6 - Personal Privacy

To: Washington, Valerie[Washington.Valerie@epa.gov]
From: Dourson, Michael
Sent: Mon 11/6/2017 12:36:16 PM
Subject: RE: Compressed Day

Valerie

Thanks for letting me know. Ex. 6 - Personal Privacy

Michael

-----Original Message-----
From: Washington, Valerie
Sent: Monday, November 6, 2017 6:21 AM
To: Wooden-Aguilar, Helena <Wooden-Aguilar.Helena@epa.gov>; Allen, Reginald <Allen.Reginald@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Greenwalt, Sarah <greenwalt.sarah@epa.gov>
Cc: Willis, Sharnett <Willis.Sharnett@epa.gov>
Subject: Compressed Day

Gm All,
I am using today for my compressed day Ex. 6 - Personal Privacy on Thursday, November 9 I will use sick leave.
Thanks have a nice day

Sent from my iPhone

To: Bolen, Derrick[bolen.derrick@epa.gov]
From: Dourson, Michael
Sent: Tue 11/14/2017 2:39:26 PM
Subject: Re: Sick

Derrick

Good luck Ex. 6 - Personal Privacy

Michael

Sent from my iPad

> On Nov 14, 2017, at 8:32 AM, Bolen, Derrick <bolen.derrick@epa.gov> wrote:
>
> All-
>
> I will be out of the office today Ex. 6 - Personal Privacy I can still answer your emails so no worries.
>
> Thank you,
> Derrick Bolen
>

To: Morris, Jeff[Morris.Jeff@epa.gov]
Cc: Beck, Nancy[Beck.Nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/25/2017 10:53:50 AM
Subject: Re: PRE-PRIORIZATION PUBLIC MEETING

Jeff

Very nice thought. I will get a sense of the edges around my time commitment and see when might be best from your perspective.

Cheers!

Mike

Sent from my iPad

> On Oct 24, 2017, at 8:48 PM, Morris, Jeff <Morris.Jeff@epa.gov> wrote:
>
> Mike,
>
> The meeting is long enough that we can work around your schedule to locate a role for you on the agenda. I think it would be very useful for senior agency leadership to emphasize collaboration among the agency and its stakeholders not just in coming with approaches for identifying candidates for prioritization but also, once that's done, to work together to put those approaches into practice and implement the chemical evaluation program mandated by the TSCA amendments. Thanks.
>
> Jeff
>
> Sent from my iPhone
>
>> On Oct 24, 2017, at 1:27 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:
>>
>> Jeff
>>
>> I am currently scheduled for a session at the Society for Risk Analysis meeting from 8:30 to 10 am on 12/11. This commitment was made well before my nomination for AA. What did you have in mind for my activity at your meeting, if anything?
>>
>> Cheers!
>>
>> Michael
>>
>> Sent from my iPad
>>
>>> On Oct 24, 2017, at 1:00 PM, Morris, Jeff <Morris.Jeff@epa.gov> wrote:
>>>
>>> Additional information will be provided.
>>> <meeting.ics>

To: Griffo, Shannon[Griffo.Shannon@epa.gov]
Cc: Fugh, Justina[Fugh.Justina@epa.gov]
From: Dourson, Michael
Sent: Thur 11/16/2017 7:03:21 PM
Subject: RE: Meeting for Ethics Follow-up Questions

Shannon

Yes, I am open now and until 2:25. Cheers!

Michael

From: Griffo, Shannon
Sent: Thursday, November 16, 2017 10:22 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Fugh, Justina <Fugh.Justina@epa.gov>
Subject: RE: Meeting for Ethics Follow-up Questions

Hi Michael,

Do you still have an open window at 2pm today? We don't mind coming to your office again (3315N). Unless we hear differently, we will drop by then.

Thanks,

Shannon

Shannon Griffo

Ethics Attorney

Office of General Counsel, Ethics

U.S. Environmental Protection Agency

(202) 564-7061

Griffo.Shannon@epa.gov

From: Dourson, Michael
Sent: Wednesday, November 15, 2017 8:09 PM
To: Griffo, Shannon <Griffo.Shannon@epa.gov>
Cc: Fugh, Justina <Fugh.Justina@epa.gov>
Subject: Re: Meeting for Ethics Follow-up Questions

Shannon

Sorry, just got your email, as I was in meetings all day. Tomorrow is not much better, but I do have a window open up at 2 pm? Your place?

Cheers!

Michael

Sent from my iPad

On Nov 15, 2017, at 11:55 AM, Griffo, Shannon <Griffo.Shannon@epa.gov> wrote:

> Hi Michael,
>
> Justina and I have some follow-up questions related to your recusal that we need to address before we can send our ethics responses to the SEPW letter. Do you have any time today to chat?

Thanks,

Shannon

Shannon Griffo

Ethics Attorney

Office of General Counsel, Ethics

U.S. Environmental Protection Agency

(202) 564-7061

Griffo.Shannon@epa.gov

To: Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]
From: Dourson, Michael
Sent: Mon 11/20/2017 6:58:18 PM
Subject: SNURs

Nancy and Charlotte

Do you need me for tomorrow's SNUR meeting at 1 pm? If not, I plan to attend the EPA PFOA meeting.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

To: Beck, Nancy[Beck.Nancy@epa.gov]
Cc: Keigwin, Richard[Keigwin.Richard@epa.gov]; Keller, Kaitlin[keller.kaitlin@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]
From: Dourson, Michael
Sent: Tue 11/28/2017 10:05:54 AM
Subject: Re: ICYMI- glyphosate

Nancy

Have our ORD colleagues been contacted for concurrence on revised OPP position?

Cheers!

Michael

Sent from my iPhone

On Nov 27, 2017, at 9:38 PM, Beck, Nancy <Beck.Nancy@epa.gov> wrote:

> https://mobile.nytimes.com/2017/11/27/business/eu-glyphosate-pesticide.html?_r=0&referer=https://www.google.com/
>
> _____
> Nancy B. Beck, Ph.D., DABT
> Deputy Assistant Administrator, OCSPP
> P: 202-564-1273
> M: Ex. 6 - Personal Privacy
> Beck.Nancy@epa.gov

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 11:10:53 PM
Subject: RE: Peer Review

Another nice note…

From: Beck, Nancy
Sent: Thursday, October 19, 2017 8:08 AM
To: Barone, Stan <Barone.Stan@epa.gov>; Zarba, Christopher <Zarba.Christopher@epa.gov>
Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>
Subject: Peer Review

Stan and Chris,

I've mentioned to you both the idea of Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Please let me know if you have any questions.

Thanks,
Nancy

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M Ex. 6 - Personal Privacy

beck.nancy@epa.gov

Cc: Beck, Nancy[Beck.Nancy@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Henry, Tala[Henry.Tala@epa.gov]
To: Morris, Jeff[Morris.Jeff@epa.gov]
From: Dourson, Michael
Sent: Tue 11/14/2017 2:38:45 PM
Subject: Fwd: PFOA
Emmett_2006_JOEM_Community exposure to PFOA.pdf
ATT00001.htm

Jeff

Thanks for the heads up on genX. I also have been sending around the attached study which

Ex. 5 - Deliberative Process

Cheers!

Michael

Sent from my iPad

Begin forwarded message:

From: "Dourson, Michael" <dourson.michael@epa.gov>
Date: November 14, 2017 at 7:53:22 AM EST
To: "Rodan, Bruce" <rodan.bruce@epa.gov>, "Flowers, Lynn" <Flowers.Lynn@epa.gov>
Cc: "Beck, Nancy" <beck.nancy@epa.gov>, "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>, "Ohanian, Edward" <Ohanian.Edward@epa.gov>
Subject: FW: PFOA

Bruce and Lynn

I understand that you both are involved with PFOA issues. Attached is the study on PFOA

Ex. 5 - Deliberative Process

The Office of Chemical Safety and Pollution Prevention senior staff and I are looking

forward to a briefing from OW on their health advisory. We would be more than happy to let you know when this is occurring, if you have not already been brought up to speed on this.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

NIH Public Access
Author Manuscript
J Occup Environ Med. Author manuscript; available in PMC 2011 February 14.

Published in final edited form as:
J Occup Environ Med. 2006 August ; 48(8): 759±770. doi:10.1097/01.jom.0000232486.07658.74.

Community Exposure to Perfluorooctanoate: Relationships Between Serum Concentrations and Exposure Sources

Edward Anthony Emmett, MD, MS[1], **Frances Susan Shofer, PhD**[1], **Hong Zhang, MD,MPH**[2], **David Freeman, MS**[3], **Chintan Desai**[1], and **Leslie Michael Shaw, PhD**[1]

[1]University of Pennsylvania, School of Medicine, Philadelphia, PA

[2]Grand Central Family Medicine, Parkersburg, WV

[3]Decatur Community Association, Cutler, OH

Abstract

Objective—To determine serum [PFOA] in residents near a fluoropolymer production facility: the contributions from air, water and occupational exposures, personal and dietary habits, and relationships to age and gender.

Methods—Questionnaire and serum PFOA measurements in a stratified random sample and volunteers residing in locations with the same residential water supply but with higher and lower potential air PFOA exposure.

Results—Serum [PFOA] greatly exceeded general population medians. Occupational exposure from production processes using PFOA and residential water had additive effects, no other occupations contributed. Serum [PFOA] depended on the source of residential drinking water, and not potential air exposure. For public water users the best-fit model included age, tap water drinks per day, servings of home-grown fruit and vegetables, and carbon filter use.

Conclusions—Residential water source was the primary determinant of serum [PFOA].

INTRODUCTION

Fluoropolymers are used in a variety of industrial and consumer products, including protective coatings for carpets and apparel, consumer housewares, paper coatings, electronics, insecticide formulations, surfactants, aerospace and other applications.

Perfluorooctanoate (PFOA, CF_3, $(CF_2)_6$ $C00^-$, CAS No 3825-26-1) has commercial use primarily as ammonium perfluorooctanoate, an essential surface-active agent in the production of various fluoropolymers, including tetrafluoroethylene. PFOA is a contaminant in other fluorochemicals and telomer products (1). According to manufacturers, it is typically not present in finished consumer articles. Ammonium perfluorooctanoate is fully dissociated into the anion form, perfluorooctanoate, in environmental media and biological fluids.

Organofluorine compounds behave very differently to the more widely studied organochlorines and organobromines and have unusual partitioning properties (2). Perfluorofatty and perfluorosulfonic acids, particularly PFOA and perfluorooctane sulfonate

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(PFOS), are now found ubiquitously in marine animals inhabiting widely spread geographical biospheres (3) and in human serum from widely disparate groups (4–7). PFOA and PFOS persist in the environment and resist biological, environmental or photochemical degradation (3M 2001). They have no known natural sources (8).

In the general US population, median serum PFOA values are around 4 to 5 ng/mL, occasional values are above 20 ng/mL (4,5,9) with no significant gender differences. Analyses of blood samples from residents near Washington County, Maryland found a 2-fold increase in serum PFOA levels between 1974 and 1989 (6). Kannan et al (7) have reported differences in blood serum PFOA levels among populations from different countries.

PFOA toxicology has recently been reviewed (1). PFOA is well absorbed by rats following both oral and inhalation exposure. Fecal excretion in male rats is increased by feeding cholestyramine resin, suggesting enterohepatic circulation (10). Dermal penetration is significant in rats but is low to negligible in humans (11). In rats, PFOA is a peroxisome proliferator activated receptor (PPAR) agonist causing liver toxicity (12,13) with hepatomegaly and hepatic necrosis, and biochemical effects characteristic of PPAR agonists (14). PFOA promotes liver carcinogenesis in rats (15), and causes Leydig-cell testicular tumors and acinar cell pancreatic tumors (16,17), through non-genotoxic mechanisms (18,19) with questionable human relevance. The human half-life of PFOA was between four and five years for retirees with previous heavy occupational exposure (20), much longer than in laboratory animals.

Control of human exposure to PFOA has been limited by the lack of information on sources and pathways. As the US Environmental Protection Agency states: "At present, there aren't any steps that EPA recommends that consumers take to reduce exposure to PFOA because the sources of PFOA in the environment and the pathways by which people are exposed are unknown. The limited geographic locations of fluorochemical plants making or using the chemical suggest that there may be additional sources of PFOA in the environment and exposures beyond those attributable to direct releases from industrial facilities. But whether human exposures are due to PFOA in the air, the water, on dusts or sediments in dietary sources or through some combination of routes is currently unknown" (21).

PFOA has been used in the manufacturing of fluoropolymers at a facility in Washington, WV since 1951. Potential airborne PFOA exposure was modeled using information on releases from the plant, meteorological conditions and topography. The wind rose-map, which shows the frequency and strength of winds from different directions, for the plant indicates the primary wind direction, toward the north/northeast, would carry airborne emissions into neighboring Ohio. PFOA was also released to the Ohio River, adjacent to the plant, as well as disposed in landfills and surface impoundments in the vicinity. According to the facility, total PFOA emissions from the facility have been reduced from 87,000 lbs (31,000 air, 56,000 water) and 80,000 lbs (31,000 air, 49,000 water) in 1999 and 2000 respectively, to 11,000 lbs (6,000 air, 5,000 water) and 1,700 lbs (200 air, 1,500 water) in 2003 and 2004 respectively.

PFOA has been detected in public and private drinking water supplies near the facility. The highest levels reported in public water supplies in the US to date have been in the Little Hocking water system, in operation since 1968, which draws water from wells across the Ohio river from the facility. The average [PFOA] in Little Hocking system distribution water for 2002–2005 has been 3.55 ng/mL (range 1.5 ng/mL to 7.2 ng/mL).

The objectives of the present study were to measure serum PFOA levels in a stratified random sample of the population served by the Little Hocking water service to determine:

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how the serum PFOA levels compared with levels measured in other populations; the relative contributions of air and water exposure to serum PFOA levels; and to determine the effects, if any, of demographic variables, occupational exposures, personal habits, use of water filters and dietary factors such as the ingestion of locally-harvested game and fish and of homegrown vegetables.

MATERIALS AND METHODS

Eligibility Criteria

Eligibility criteria for participation in the study were:

- Residence in the area serviced by the Little Hocking Water Association for at least the past two years, as of July 2004
- Ages two or older (changed to ages four or older after the study commenced to minimize participant discomfort) and
- Not known to have a bleeding disorder (in order to diminish any risk from phlebotomy).

Selection of Households for Sampling Frame

Two populations of residents were identified for participation in the stratified random sampling. One population represented those whose residence was potentially exposed to PFOA in both air and water, the other whose residence was potentially exposed to PFOA in water but had very minimal potential for exposure in air. The sampling randomly selected households from each of these strata.

To identify areas where there was higher exposure to PFOA in the air, we used an air dispersion model that estimated the air concentration for PFOA emanating from the PFOA source plant. Inputs into the air dispersion model included the amounts of air emissions for the plant, wind velocities, and topographic contours. The air concentrations had been modeled for years 2002 & 2003 on an annual basis; the model produced very similar results for each of these years. To identify areas in the Little Hocking water service distribution area, a map of the water distribution system was obtained for the Little Hocking water service. The potential air and water exposure group comprised all those who had resided for at least two years in the water distribution system area of the Little Hocking water service and also within the contour line representing 0.2 $\mu g/m^3$ PFOA in the air as a yearly average for 2002. These households were all located in portions of Zip Codes 45714 (Belpre) and 45742 (Little Hocking).

The potential water exposure group comprised residents who had resided for at least two years in the water distribution system area of the Little Hocking water service but in an area where air exposure to PFOA from the facility was negligible. The selected study area was zip codes 45724 (Cutler), and 45784 (Vincent). These areas were all at least several miles outside the lowest air concentration contours derived from the air dispersion model. Figure 1 shows the location of the residence areas for both the potential air and water exposure and the potential water only exposure zones.

To identify households and residents in the zip codes of interest, demographic and other information was purchased from www.infousa, a proprietary database of detailed information on US consumer households compiled from thousands of public sources. The items used to select invitees were names of head of household, street address, city, state, ZIP Code, and length of residence.

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Selection of Stratified Random Sample—For the area identified as having both air and water exposure 95 households in the www.infousa database met the requirements, all were invited to take part in the study. These included households with measured PFOA levels in potable well-water, measured by the Ohio Department of Environmental Protection and households using Little Hocking Water Association water. For the area identified as having only water exposure to PFOA, a stratified random sampling of households was performed, resulting in the selection of 342 households. All members of selected households who met the study eligibility criteria were invited to participate.

Invitations to participate—Invitation letters were sent from the University of Pennsylvania to each selected household. If no response was received, a second mailing was sent. If there was still no response after approximately 10 days, a telephone call was made to the household by staff of the Decatur Community Association. No participant chose an option for anonymous participation. On the weekend prior to the mailing of the invitation letter, a flyer was placed in the area weekend newspaper to announce that invitation letters were forthcoming. The principal local newspaper, the Marietta Times, independently wrote an editorial encouraging those selected to consider participation.

Community Volunteer Group—Because of great community interest, a lottery was conducted to select an additional sample of invitees from households that volunteered to participate in the study in response to a newsletter notice. Those households that met study criteria including residing in one of the areas used for stratified random sampling were included in the lottery.

Administration of Questionnaires

Administration of questionnaires and collection of blood samples were performed between July 2004 and February 2005, in nearby Parkersburg, WV. The questionnaires were developed and revised after review by the members of the Community Advisory Committee and an expert panel from the US EPA. The Community Advisory Committee, convened by the Decatur Community Association, comprised representatives of the townships in the Little Hocking Water Association Service District, representatives from the Ohio and US EPA, the Warren School District and the County Health Commissioner. Prior to finalization, the questionnaires were pilot tested on a representative group of 20 individuals from similar Southeastern Ohio or Western West Virginia communities, who did not live in the Little Hocking Water Association District.

Trained interviewers administered all questionnaires. Only one person from each household supplied household information. The household questionnaire elicited information to ensure that participants met the eligibility criteria, demographic information on eligible participants, household contact information, and sources of residential drinking water [private well, water district, cisterns, bottled water, hauled water, etc.], use of a home water filter, and water source and estimated usage for cooking, canning, and reconstituting canned soups and frozen juices.

All adults 18 years and older were administered the adult questionnaire that elicited demographic information, diet (including consumption of vegetables or fruit grown in your garden, meat or game grown locally, and fish caught locally), health conditions (liver, thyroid, bleeding disorders), current medications, current occupational or school if a full-time student, employment (including at a facility using PFOA, visiting or processing waste from that facility, work as a firefighter, in carpet cleaning or retreating carpets or rugs, or in professional carpet installation), and smoking and alcohol habits.

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All children were administered a questionnaire that was similar to the adult questionnaire except that the questions about occupation and about smoking and alcohol habits were omitted.

Collection and Assay of PFOA Acid in Serum

Specimen collection—Twenty mls of blood were drawn into red-topped Vacutainer tube for PFOA analysis, immediately centrifuged, and the resulting serum was transferred to polypropylene aliquot tubes, labeled and shipped on dry ice to the analysis laboratory (Exygen Research) where it was stored at −80 °C pending analysis.

Standards and chemicals—The standard for perfluorooctanoic acid (99.2%) was obtained from Oakwood Products, Inc (West Columbia, SC) and characterized by DuPont (Newark, DE). Analysis by ^{19}F NMR confirmed that the PFOA standard contained 98.7% straight chain PFOA and 0.53% branched PFOA isomers. The internal standard, [1,2-^{13}C]-PFOA($C_6F_{13}CF_2{}^{13}CO_2H$, ^{13}C-PFOA) (96.4%) was provided by DuPont (Newark, DE).

Chemicals and reagents used in the sample preparation procedure or in the mobile phase were of reagent grade and were obtained from VWR Scientific (Bridgeport, NJ) and Sigma-Aldrich (St Louis, MO). Solvents used for the mobile phase (acetonitrile, water) were of HPLC grade and were obtained from EM Science (Gibbstown, NJ). The control human serum was purchased from Lampire Biological Laboratories, Inc (Pipersville, PA) and stored frozen at −20 °C. This fluid was used for the preparation of laboratory quality control samples with spiked-in PFOA.

Chromatographic and Mass spectrometric conditions—PFOA was analyzed through HPLC/tandem mass spectrometry by a slight modification of the method of Flaherty et al (22).

Standards, sample preparation and calibration—Controls and study subject samples were added 300 μL of acetonitrile. The samples were thoroughly mixed by vortexing, centrifuged and 5 μL of the cell- and protein-free supernatant used for analysis by the HPLC tandem mass spectrometer system. A 7-point calibration curve was analyzed throughout the analytical sequence for the fluorocompounds. The calibrators included normal human serum spiked with 0.5, 1, 5, 10, 20, 50, and 100 ng/mL of PFOA. The instrument response versus the calibrator concentration was plotted for each point. Linear regression with 1/x weighting was used to determine the slope, y-intercept and coefficient of determination (r^2). Calibration curves were deemed acceptable if $r^2 \geq 0.985$. This is the external standardization method used for the determination of PFOA in the set of 408 samples described in this study. For samples with PFOA concentrations >100 ng/mL, the sample was diluted in 50:50 methanol/water and re-run. In addition the analysis of PFOA was done using ^{13}C-perfluorooctanoic acid as an internal standard for a randomly selected set of 35 of the samples in order to certify that the external standardization method used provided equivalent PFOA concentration values. For these analyses the internal standard was mixed in acetonitrile at a concentration of 1 ng/mL. As described above for the externally standardized assay for sample preparation: to 100 μL of standards, controls and study subject samples was added 300 mL of acetonitrile containing the internal standard and the cell-and protein-free supernatants prepared as above. On comparison of the externally standardized with the internally standardized sets of results on the 35 selected samples, linear regression analysis showed excellent agreement between the two calibration procedures: $Y(IS) = 1.073 \pm 0.0229 * X(\text{ext std}) - 0.385 \pm 0.468$; $r^2 = 0.985$; $S_{y \cdot x} = 1.54$.

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Matrix spike samples and duplicate sample assays—One matrix spike for every 20 samples was prepared by adding a known concentration of the PFOA to the study subject serum sample for the purpose of assessment of the method's accuracy throughout the set of study subject serum samples. The mean PFOA recovery for these spiked samples was 95% with an SD of 16.2%. In addition, one sample of every 10 was extracted and analyzed in duplicate in order to provide an assessment of the method's precision throughout the set of samples. The average between assay %CV for PFOA duplicates was 5.7%. The lower limit of quantification of this method is 0.5 ng/mL. Validation of this LLOQ was conducted with replicate spiked samples of human serum with PFOA spiked into the samples at 0.5 ng/mL, the concentration of the lowest calibrator for this assay. The mean recovery + SD was 101 + 2.7%.

Serum [PFOA] Philadelphia Volunteer Group—To help ensure that published general population serum PFOA levels were suitable for comparison purposes under the circumstances of the study, we identified a comparison group of 30 volunteers from the Philadelphia area. The Philadelphia volunteers, staff and students at the Hospital of the University of Pennsylvania, were paid $20 each to participate. Their mean age was 34.3, range 20–56; there were 9 men and 21 women. None identified previous or current occupational exposure to PFOA. Blood from these individuals was drawn, handled spun, stored, shipped and analyzed for PFOA in an identical manner to the blood obtained during the study. The mean serum PFOA levels for the Philadelphia comparison group was 6 ng/mL, IQR 5–10 ng/mL consistent with published values for the US population (4,5,6).

[PFOA] Water Sampling and comparison to serum levels

The concentration of PFOA in finished water in the Little Hocking water system has been measured approximately quarterly from 1/22/2002 to 5/18/2005 by the Ohio EPA. Fourteen measurements were available for this period, results before 11/29/04 had been reported as ammonium perfluorooctanate (APFO), and as PFOA from that date. PFOA concentration in private residential well water was publicly available for 9 individuals for whom private well water was their only reported source of residential drinking water. In one instance, 6 samples had been taken at regular intervals from 2002 through 2005. For this well, the values obtained were averaged to obtain a mean level over the period. For the remaining wells only one sample had been analyzed from a single point in time. The average PFOA concentration in Little Hocking system distribution water from January 2002 until May 2005 was 3.55 ng/mL (range 1.5 ng/mL to 7.2 ng/mL). For private wells used by study participants, PFOA concentrations ranged from not detectable (<0.010 ng/mL) to 14.0 ng/mL.

Statistical Analysis

To determine if serum PFOA levels differed by dietary or personal habits, water source, water usage, occupational exposure, etc., preliminary data analyses included the t-test for binary predictors or the analysis of variance (ANOVA) for greater than 2 exposure categories. Adjustment for multiple comparisons were made using Tukey-Kramer. To check the assumptions of the statistical approach used, various analyses were rerun with the exact test using Monte Carlo. Results were similar to that of the f test. Subsequent higher order analyses included analysis of covariance adjusting for age. Final multivariate analysis to assess the independent contribution of multiple variables was a generalized estimating equation (GEE) to adjust for household cluster. Only variables associated with serum PFOA levels on univariate analysis with a probability <.10 were included. To determine model of best fit, both forced entry and backward elimination were employed. All analyses were performed using SAS statistical software (Version 9.1, SAS Institute, Cary NC). A $p<.05$

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was considered statistically significant. Serum PFOA levels Serum [PFOA] are presented as mean, median, and interquartile range (IQR).

To examine the effect of demographic variables (age, gender, duration lived at current residence) we excluded the 18 participants who reported substantial occupational exposure (defined below) to PFOA. To examine the effects of number of glasses of drinking water per day, use of a residential water filter and of dietary exposures we included only those residents whose sole source of residential drinking water was Little Hocking water system water. Only individuals who designated a single source of residential drinking water, and who did not have substantial occupational exposure to PFOA were included in these analyses.

Human Subjects Approvals

The study was approved by the Institutional Review Board (IRB) of the University of Pennsylvania. The study was voluntary and informed consent was obtained for all participants prior to any study. Minors under the ages of 17 were encouraged to give informed assent whenever feasible. A Certificate of Confidentiality was obtained from the NIH to ensure maximum protection of personal information and results.

A partnership between the University of Pennsylvania School of Medicine, The Decatur Community Association, a local community association in the Little Hocking water service area, and Grand Central Family Medicine in Parkersburg WV, a local health care provider, conducted the study through a grant from the Environmental Justice Program of NIEHS. The community was involved at all stages of the study. A local health-care provider informed each participant of his or her personal PFOA results together with any necessary explanation.

RESULTS

Response and Participation Rate

Stratified Random Sample—343 individuals from 169 households participated in the phlebotomy and questionnaire administration. One subject withdrew from the study, 6 subjects could not donate sufficient blood, one subject did not complete the questionnaire, and 11 subjects did not meet eligibility criteria because their household water service was received from a water system other than the Little Hocking Water Association. Accordingly, data was available for analysis from 324 subjects from 161 households selected through the stratified random selection process. The participation rate by location of household mailing address is given in Table 1.

Response and Participation - Community Volunteer Group—100% of the 37 households selected by lottery participated in the phlebotomy. However, 2 individuals from 2 households did not complete the questionnaire and were excluded from further analysis. Thus data from 54 individuals from 35 households was included in the final analysis. The racial and ethnic composition of both participants and volunteers was predominantly white non-Hispanic (97%, N=367), reflecting the composition of Washington County, Ohio.

Role of Occupational Exposure

We established criteria for substantial occupational exposure to PFOA of: at least one years' work in a production area within a facility in which PFOA was used in the production process; with the last such occupational exposure within the previous 10 years. Seventeen individuals from the stratified random sample, and one from the local volunteer sample met this definition for substantial occupational exposure. All had received their occupational

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exposure to PFOA in the same fluoropolymer manufacturing facility located in Washington, WV across the Ohio River from the study area. An additional 48 individuals reported past or current potential occupational exposure to PFOA as follows (individuals can be represented more than once): 18 individuals had worked in a fluoropolymer manufacturing facility in a non-production area, at the fluoropolymer production facility in a production area for less than one year total and/or more than ten years ago, or in a job for another employer that required visits to the fluoropolymer production facility, so did not meet the criteria for substantial occupational exposure; 8 individuals had worked in a job involving waste disposal or waste processing from the fluoropolymer manufacturing facility; 29 individuals had worked as firefighters (volunteer, military, as a company employee or paid) and 13 individuals had worked in carpet cleaning, retreating carpets or rugs, or in professional carpet installation. Compared to the no exposure group, none of these occupational exposure groups had statistically significant elevated serum PFOA levels ($p>.05$) (Table 2). Among those with potential occupational exposure, the highest median values were observed for firefighters. However, these values remained well below the concentrations of the substantial occupational exposure group. Since none of these groups had significantly elevated serum PFOA levels they were aggregated into one group (potential exposure) for statistical analysis purposes.

When comparing substantial, potential, and no occupational exposure groups, the substantial occupational exposure group had a significantly higher median serum PFOA levels of 775 ng/mL than the potential exposure (388 ng/mL), and no occupational exposure groups (329 ng/mL) ($p=.0002$, $p<.0001$ respectively, Table 2).

As a result of this finding, the substantial occupational exposure group was removed from further analysis of PFOA exposure in the community. Since the serum PFOA levels for the potential exposure group were not different from the rest of the community, they were included in subsequent analyses of community exposures and treated for purposes of analysis as residents without substantial occupational exposure.

Role of Community Air Exposure: Serum [PFOA] by Community of Residence

The median serum PFOA level in the combined two areas with highest potential air exposure (Little Hocking and Belpre) was 326 ng/mL, compared to 368 ng/mL in the combined two areas with a potentially minimal contribution from PFOA through air pollution (Cutler and Vincent) (Table 3). This difference was not statistically significant ($p=.32$).

Additionally, the inclusion of local volunteers made no appreciable difference to the results (Table 3). Because of the similarity of serum PFOA levels in each community regardless of air pollution or the inclusion of volunteers, all communities and samples were combined in the subsequent analyses to examine the effects of water exposure on [PFOA].

Role of Exposure in Water: Serum [PFOA] and Primary Source of Residential Drinking Water

With regard to water exposure, the highest median serum PFOA level (374 ng/mL) was found for the group who used only Little Hocking system water as their residential drinking water source (Table 4). The lowest was found in those who currently used only bottled and/or cistern and/or spring water as the source of their residential drinking water. The serum PFOA levels in those who used bottled, spring or cistern water was significantly lower than those in both the Little Hocking water system only and the mixed Little Hocking plus another water source groups ($p= .0004$, and $p= .007$ respectively. The serum PFOA levels

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for those who used Little Hocking water system water only and the mixed Little Hocking and another water source were not statistically significantly different (p=.17).

The mean serum PFOA levels in those who used any well water as their sole residential drinking water source was variable; this group included some of the lowest and some of the highest PFOA serum concentrations.

Relationship between [PFOA] in primary residential water supply and serum [PFOA] in residents—Figure 2 presents a graphical relationship between PFOA concentrations in drinking water and serum PFOA levels. Three individuals drank from wells where the PFOA was not detectable, their average serum PFOA level was 20.8 ng/mL, (range 13.6 to 31.4 ng/mL). Six individuals used a private well with measurable PFOA in water as their only source of residential drinking water. Although the numbers of individuals for whom the PFOA concentration in well water is known is small, there is an apparent strong relationship between the level of the serum PFOA levels and the PFOA concentration of the drinking water source.

The median serum/drinking PFOA water ratio residents using only the Little Hocking water system was 105 (371/3.55), with an interquartile range between 62 (221/3.55) and 162 (576/3.55). For the six individuals who used a private well with measured [PFOA] as their only source of residential drinking water, the serum/drinking water PFOA ratios ranged from 142 to 855.

Serum PFOA levels and gender, age, years of residence, smoking and alcohol

Serum PFOA level was not significantly different by gender for participants without substantial occupational exposure (p=.32). The median [PFOA] for females was 320 ng/mL, IQR 161–509, and for males was 345, IQR 190 to 576.

Serum PFOA concentrations were highest in those aged more than 60, followed by those aged from 2–5, and those aged 51–60 (Figure 3). Participants >60 years were significantly more likely to have higher serum PFOA levels compared to participants in all other age groups except children 2–5 years old (.0006< p <.02).

With regard to residence, only participants over 18 years were examined. Years lived at current residence was grouped into 2–5 years, 6–10 years, 11–15 years, and >15 years. Age was also found to be correlated with years of residence (r= .6). Therefore, age was controlled for in the analysis for which no statistically significant association between years lived at current residence and serum PFOA levels was found (p=0.7).

The influence of alcohol consumption (consumption of beer wine or liquor in last thirty days) and smoking (current cigarette smoker) were evaluated in all adult participants ages 18 and over who did not have substantial occupational exposure. No significant association was found between serum PFOA levels and smoking (p=0.28) or serum PFOA levels and alcohol consumption (p=0.46)

Little Hocking Water System Users: Water Usage Variables Affecting Serum PFOA Concentrations

The effect of drinking tap water, eating local fruits and vegetables, meat or fish, or having a carbon water filter on serum PFOA concentrations in Little Hocking Water System Users is shown in Table 5. With increasing tap water drinks per day (at home or at work), PFOA levels increased (p=.004). Particularly, participants who drank 8 or more cups of tap water per day (at home or at work) had significantly higher serum PFOA levels compared to other drinking categories (.002 < p < .004).

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A secondary analysis has been performed, examining air exposure and local vegetable/fruit intake. There was no effect of air exposure on PFOA (p=.16) or the interaction between air exposure and local vegetable/fruit intake (p=.73) As a result of the lack of association between these 2 variables, air exposure was not included in the GEE model. Similarly, there was a statistically significant increase (p=0.0002) in the mean serum [PFOA] associated with increasing numbers of weekly servings of fruits and vegetables from a local garden. Additionally, there was an increase in serum PFOA with servings of meat or game grown or harvested locally (p=.005). No association was found between local fish consumption and serum PFOA concentrations.

With regard to water filtration systems, residents using only Little Hocking water system water as their residential drinking water source were divided into 2 groups: those using a home water filter system based on carbon (N=64) and those who had no home water filtration system or used a system not known to remove PFOA, or used a system whose type and composition could not be verified (N=209). Residents using carbon water filters had significantly lower median serum PFOA levels (318 ng/mL), compared with residents using Little Hocking System water who did not use carbon water filtration (421 ng/mL) (p= .008)

Serum PFOA levels and Household Cooking Use of Tap Water

There was no relationship between serum [PFOA] and the use of tap water in cooking for those households using only Little Hocking water system water (Figure 4). When cooking vegetables and pasta, making soups and stews, reconstituting canned soups, reconstituting frozen fruit juices and home canning of vegetables and meats were examined, no statistically significant relationship with serum PFOA levels was found. However a linear trend of increasing serum PFOA levels was observed with increasing use of water for making soups and stews and for home canning of vegetables and meats.

Little Hocking Water System Users: Multivariate Analysis Adjusting for Household Clustering

The model of best-fit included age, tap water drinks per day, fruit and vegetable servings per week from your garden, and use of a carbon filter (Table 6). Eating meat and game grown or harvested locally was not found to be associated with serum PFOA levels in the multivariate analysis.

DISCUSSION

We found that median serum PFOA levels in randomly selected residents of the Little Hocking water service district ranged from 298 to 370 ng/mL, in the order of 60 to 75 times the median levels of approximately 5 ng/mL previously described for general US populations (4,5,6). The majority of serum PFOA levels in these residents exceeded the maximums reported in previous community studies in other geographic locations. For example, the range of serum PFOA levels for 645 U.S. adult blood donors was from 1.9 ng/mL to 52.3 ng/mL (4), for 238 elderly volunteers in Seattle was 1.4 ng/mL to 16.7 ng/mL (5) and for 598 children from across the US was from 1.9 ng/mL to 56.1 ng/mL (9). The serum PFOA levels for the thirty comparison subjects for the Philadelphia area in our study all fell within previously reported normal population ranges.

Our random sampling of residents in the water district included a number of individuals who worked in the production area of a fluoropolymer manufacturing facility located across the Ohio River in Washington, WV. This facility is believed to be the primary source of PFOA pollution in the area. A recent study of workers at this plant found the median serum PFOA level of 490 ng/mL for 259 workers currently working in production areas where PFOA was

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used (23). We found a median serum PFOA level of 774 ng/mL for the 18 workers who had worked in the production area at the facility, lived in the Little Hocking water service area, and participated in our study. The median serum PFOA level for these 18 individuals was 284 ng/mL higher than the median reported for all production workers at the facility, suggesting a combination of residential water and occupational contributions to the PFOA body burden. Since all but one of the production workers we studied was selected through stratified random sampling, we consider it unlikely that selection bias could explain this elevation. Workers from non-production areas of the facility included in our sampling did not have significantly increased serum PFOA levels compared with other residents. The serum PFOA levels in non-occupationally exposed community residents in the Little Hocking water service district approached and frequently surpassed those measured in production workers exposed to PFOA at the source fluoropolymer manufacturing plant. These results illustrate that body burdens of pollutants sustained through community environmental exposures are not necessarily less than those sustained through occupational exposure.

We were able to explore other potential occupational exposure contributions to the serum PFOA levels. In addition to use in the manufacture of fluoropolymers, it has been suspected that PFOA may also be a breakdown product of fluorinated telomers. PFOA is used as a surfactant or surface treatment chemical in many products, including fire-fighting foams; personal care and cleaning products; oil, stain, grease and water repellent coatings on carpet; textile leather and paper (21). PFOA has had limited use as a fire suppressant. A study of PFOA in consumer products identified extractable PFOA in carpet-care solution treated carpeting (24). Because PFOA and related fluorinated compounds are currently unregulated, there is relatively little available information on the extent of their use. Based on a qualitative assessment of potential occupational exposure to PFOA in the Southeastern Ohio area, we explored occupational exposure in firefighting, carpet cleaning and carpet installation in addition to potential exposure in the disposal or incineration of PFOA and/or waste from the fluoropolymer manufacturing facility. We did not observe a significant increase in median serum PFOA concentration in any of these occupational groups. It remains possible that in a population with less exposure to PFOA from ambient contamination, and identifiable contributions to the body burden might be found from one or more of these occupational exposures.

Several observations support the conclusion that the major source of the PFOA in Little Hocking water district residents was drinking water. Serum PFOA levels were similar whether residents lived in the area proximate to the plant where the air plume would have been concentrated, or in an area which had the same water service but was located up to 20 miles from the plant and where air pollution with PFOA was estimated to be minimal. Serum PFOA levels were considerably lower in those residents who were currently using only bottled, spring, or cistern water as their drinking water source. Where the primary drinking water source was well water, serum PFOA levels varied in proportion with well water PFOA levels.

The median serum/drinking water PFOA ratio of 105 we observed in Little Hocking water users likely reflects both high PFOA absorption after oral ingestion and a long half-life of PFOA in human blood. In rats, the oral bioavailability of PFOA is approximately 100% (25). The serum half-life varies widely by species and sex: several hours for female rats, about 7 to 10 days for male rats (25): 20.9 days for male and 32.6 days for female cynomolgus monkeys (26). The half-life in humans appears to be much longer. In the one set of data that is available, a study of 9 retirees from a fluoropolymer production facility, the mean serum PFOA half-life was found to be 4.4 years (20). However, we did not find a relationship between serum PFOA levels and length of residence in the Little Hocking water

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district among study participants, all of whom had lived in the area for at least two years. If the half-life in the general community is in the order of 4 to 5 years we would have expected to find a significant relationship with duration of residence. Our results thus lead us to question whether the serum PFOA half-life in the general community is as long as that published for the small retired worker group (20). We expect to have more data on this subject from a follow-up study.

In residents who drank only Little Hocking system water the model of best-fit for serum PFOA levels included age, tap water drinks per day, fruit and vegetable servings per week from a local garden, and use of a carbon water filter. The finding that PFOA concentrations were higher in children aged 5 and below and in the elderly aged over 60 is disturbing, since these may represent groups particularly vulnerable to adverse health consequences (27,28). The reason for the higher serum PFOA levels in those aged 60 and above is not entirely clear, multivariate analysis shows the increased consumption of drinking water in this group does not fully explain the observed increase. Both the elderly and those aged 5 and below may spend more time at home with exclusive use of residential water than working or school-age residents. Infants and young children may have proportionately greater exposure to water-borne pollutants since they drink more water per kg of body weight than do adults (28). The levels in the very young may also represent additional exposures as PFOA has been shown to cross the placenta and to be present in breast milk (at approximately 1/10 of the serum concentration) in Sprague Dawley rats (29), although comparable studies in humans are lacking. We are performing further studies to elucidate PFOA exposures in maternal milk and infant formula. A higher serum PFOA level for young children was previously observed by Olsen et al (9) who measured PFOA in the serum of 598 children aged 2–12 who participated in a nationwide US study of Group A Streptococcal infections, 645 adult blood donors from 6 US blood bank donation sites, and 238 elderly subjects in Seattle participating in a study of cognitive function. The geometric mean serum PFOA levels (4.6 ng/mL, 4.2 ng/mL, 4.9 ng/mL respectively) were similar in all groups. However in the children there was a statistically significant negative association with age, with the highest mean serum PFOA levels noted at age 4 and the lowest at age 12. Our failure to find gender differences is consistent with previous observations in the US general population.

The association with the number of servings of fruits and vegetables from the home garden was unexpected. Possible explanations include the use of PFOA containing water for cooking, canning and washing fruits and vegetables, PFOA in the raw fruits and vegetables, and different dietary and drinking habits in those who consume more homegrown fruits and vegetables. We consider it unlikely that PFOA is elevated in raw fruits and vegetables from the garden because as a result of the natural rainfall characteristics it is unusual to water gardens and fruit trees extensively with residential water in this district. Also the association between serum PFOA and servings of fruits and vegetables was not reduced by adjusting for residence in the areas with known higher airborne and soil levels of PFOA. We are undertaking further studies to better understand the observed association.

Individuals using carbon-type water filters for residential drinking water had a reduction of approximately 25% in median serum PFOA levels compared with those not using a filter. This reduction was much less than we have seen for those who drank only bottled, spring or cistern water. Because of limited effectiveness, potential reliability problems associated with the need to maintain the filter system, and potential health problems associated with the use of home filtration systems we do not recommend reliance on home filters to remove PFOA. New water filtration products to remove PFOA are currently being pilot tested, with prospects of wider use in the near future.

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The high serum PFOA levels in our study as a result of the relatively high exposure in drinking water, may have limited our ability to detect relatively small increases associated with contributions from ambient air pollution. Thus we cannot exclude the possibility that exposure to PFOA in air could lead to a detectable contribution to the PFOA body burden in other populations with minimal water exposure.

Our finding that the major source of serum PFOA was residential drinking water has helped empower those in the community who may choose to lower their PFOA exposure, with a view to lowering their body burden. As a result of our preliminary findings that the levels of PFOA were abnormally high in residents of the Little Hocking water district, and that the major non-occupational PFOA source was residential drinking water, the option of free bottled drinking water has been made available through the Little Hocking Water Association to those with this water service. More than half of the residents are already taking advantage of this offer. In addition, a new water filtration system designed to remove PFOA is now planned. We would anticipate that these actions should result in reduced serum PFOA levels. We plan to monitor changes in serum PFOA levels in the study group over the next eighteen months, to determine the extent of any serum PFOA reductions.

Identification of water as the major route of community exposure to PFOA in this population should encourage efforts to define exposure sources in other populations, and should provide a basis for personal and regulatory efforts to reduce human exposure to a pollutant which is of concern because of remarkable persistence in both the environment and in humans.

Acknowledgments

Sources of Support

This study was supported by grant ES12591 from the Environmental Justice Program of the US National Institute for Environmental Health Sciences (NIEHS), National Institutes of Health, and by P30 Core Center grant ES 013508 from the NIEHS.

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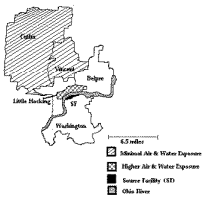

Figure 1. Map showing the locations of the studied communities and the source facility
Subjects for the minimal air exposure group were selected from the area shown in yellow, subjects for the higher air exposure group from the area shown in red. Residents in both of these areas obtained their water from the same public residential water supply. The location of the source facility is shown in black. The residents lived in Ohio, the source facility is located in West Virginia. The state boundary, the Ohio River, is shown in blue.

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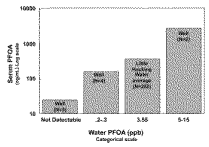

Figure 2. Relationship of PFOA Concentration in Water Source (Little Hocking & Private Wells) to Serum PFOA Levels

The numbers in parenthesis indicate the number of samples. Although the number of observations from persons using only residential well-water source is small, there is a marked and statistically significant relationship between the PFOA levels in serum and the PFOA concentration in the residential drinking water source. Only subjects 6 years of age or older using a single residential drinking water source were included in the analysis.

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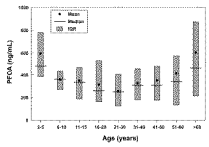

Figure 3. Distribution of Serum PFOA Levels in ng/mL by age
Residents >60 years had significantly higher serum PFOA levels compared to all other age groups except children age 2–5 years old

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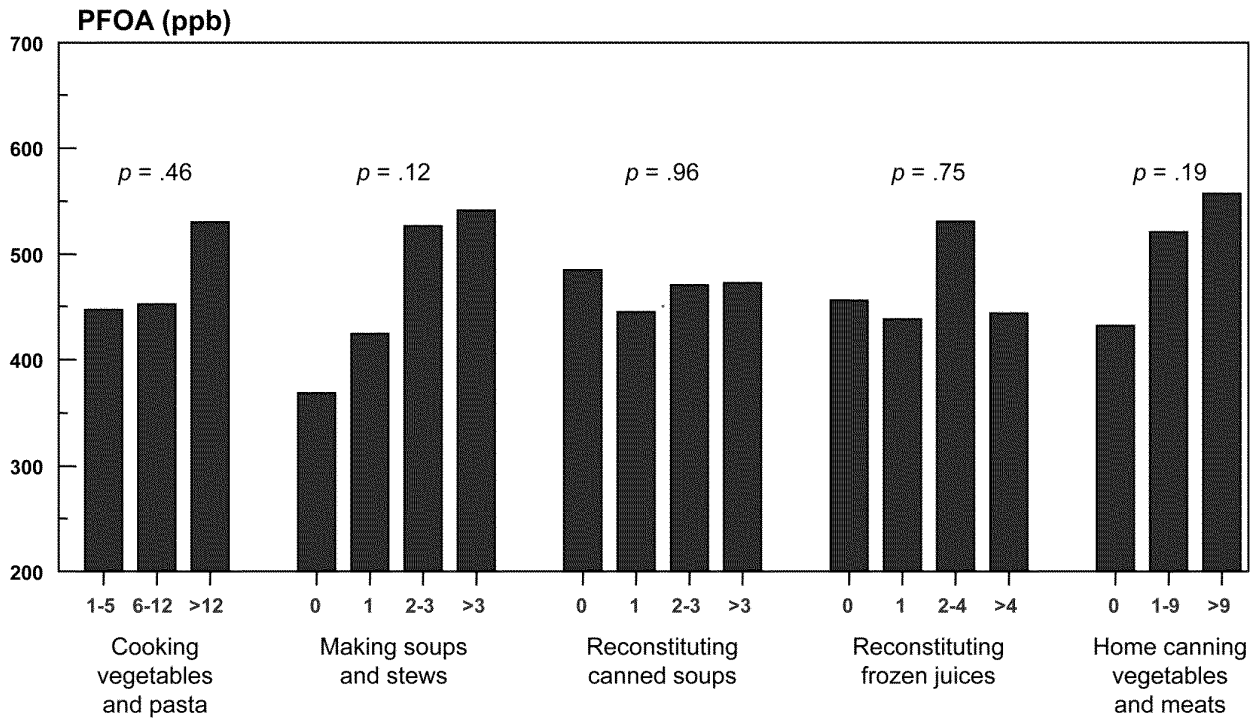

Figure 4. Distribution of serum PFOA levels in ng/mL, within household[a] for cooking tap water usage[b] (Amounts are servings per week)

[a] PFOA levels represents average household value

[b] Households using Little Hocking water system only

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Table 1

Household Participation Rates for Randomly Selected Households by Community.

	Households Invited to Participate	# Agreeing to Participate	# Completing Data Acquisition	Participation Rate
Little Hocking	78	45	38	48.7
Belpre	17	8	7	41.2
Cutler	101	45	30	29.7
Vincent	241	115	86	35.7
TOTAL	437	213	161	36.8

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Table 2

Serum [PFOA] ng/mL by Occupational Exposure Group

Occupational Exposure	N	Median	Mean	IQR
NO OCCUPATIONAL EXPOSURE	312	329	423	175–537
POTENTIAL OCCUPATIONAL EXPOSURES[a]	48	388	406	168–623
Firefighter: voluntary, military, company employee or paid	29	447	453	236–709
Non-production area of fluoropolymer facility, in production area not meeting criteria for substantial occupational exposure, or requiring visits to facility.	18	381	386	125–430
Carpet cleaning, retreating carpets or rugs, or in professional carpet installation	13	302	408	191–631
Facility processing or disposing fluoropolymer production waste	8	253	578	115–918
SUBSTANTIAL OCCUPATIONAL EXPOSURE [Production area within a facility in which PFOA was used in the production process >1 year and last exposure having occurred within previous 10 years]	18	775	824	422–999

[a]Some individuals had more than one potential occupational exposure, therefore N for the potential occupational exposure subgroups does not total to 48.

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J Occup Environ Med. Author manuscript; available in PMC 2011 February 14.

Table 3

Serum [PFOA] in ng/ml by community area, for randomly selected participants and for all participants[a].

Community Areas with Higher Expected Contribution from Air	Randomly Selected Participants				All Participants (local volunteers and randomly selected)			
	N	Mean	Median	IQR	N	Mean	Median	IQR
Belpre	14	321	298	83–533	30	307	244	103–445
Little Hocking	74	478	327	187–572	92	458	311	175–567
TOTAL	88	453	326	176–568	122	421	298	155–556
Community Areas with Minimal Expected Contribution from Air								
	N	Mean	Median	IQR	N	Mean	Median	IQR
Cutler	59	361	316	169–477	70	380	314	185–477
Vincent	160	439	370	190–570	168	438	370	188–577
TOTAL	219	418	368	182–555	238	421	361	186–555

[a] 18 subjects with substantial occupational exposure were excluded from analysis.

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Table 4

Serum [PFOA] in ng/ml by primary residential source of drinking water[a,b]. All Participants (randomly selected and local volunteers)

Drinking Water Source	N	Median	Mean	IQR	Range
Little Hocking system water only	291	374	448	221–576	7–1950
Little Hocking system plus bottled or spring	26	320	358	206–370	72–1280
Bottled and/or cistern and/or spring only*	10	71	154	49–217	12–527
Well water and well & other	26	79	296	28–155	8–4520

[a] Subjects with substantial occupational exposure to PFOA were excluded from these analyses

[b] 7 subjects did not indicate residential source of drinking water

* Significantly different from Little Hocking water only (p=.003) and Little Hocking system plus bottled or spring water (p=.05)

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Table 5

Serum [PFOA] ng/mL, number of tap water drinks per day, consumption of local meat and game, fish, vegetables and fruits and use of carbon water filter[a]

Factor		N	Mean[b]	Median	IQR	pr > t
Tap water drinks/day	0	20	374	301	233–423	<.0001
	1–2	40	324	265	176–438	
	3–4	66	413	370	206–550	
	5–8	90	450	373	242–373	
	>8	55	565	486	294–486	
Local Meat	0	157	389	329	179–498	0.018
	1–20	49	488	451	246–690	
	>20	77	516	424	295–595	
Local Fish	No	273	448	374	221–571	0.8958
	Yes	18	458	398	290–681	
Fruit and vegetables from your garden	0	133	356	295	174–485	<.0001
	1–20	75	458	420	264–661	
	>20	77	571	469	308–802	
Carbon Water Filter[c]	Yes	64	360	318	170–482	0.0005
	No	209	493	421	258–631	

[a] Little Hocking water source only

[b] Means adjusted for age unless otherwise indicated

[c] Not adjusted for age

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Table 6

Results of Application of General Estimating Equations (GEE)

Parameter	Estimate	Standard Error	95% Confidence Limits		Z	Pr > |Z|
Intercept	110.54	58.10	−3.34	224.42	1.9	0.0571
Vegetable and fruit from your garden servings/week	62.31	20.96	21.23	103.39	2.97	0.0029
Tap water drinks/day	5.93	2.02	1.97	9.88	2.94	0.0033
Age (yrs)	3.53	1.03	1.50	5.55	3.42	0.0006
No carbon filter use	104.92	35.86	34.65	175.20	2.93	0.0034

Note: This analysis includes only participants from households using Little Hocking water system only. Participants with substantial occupational exposure were excluded

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To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 11/15/2017 1:22:36 PM
Subject: RE: Glyphosate AHS publication

Nancy

They appeared to not consider multiple comparisons based on a comment they made on page 7 of 8, second column, second paragraph, specifically,

Second, because we evaluated many cancer sites for potential associations with glyphosate use, we

Ex. 5 - Deliberative Process

Cheers!

Mike

From: Beck, Nancy
Sent: Tuesday, November 14, 2017 9:01 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: Glyphosate AHS publication

Is the methods section not sufficiently clear so that we cant tell what they did?

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

From: Dourson, Michael
Sent: Tuesday, November 14, 2017 8:53 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Glyphosate AHS publication

Nancy

Nice. But the natural question is Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Cheers!

Mike

From: Beck, Nancy
Sent: Tuesday, November 14, 2017 8:46 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: FW: Glyphosate AHS publication

FYI

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

From: Keller, Kaitlin
Sent: Saturday, November 11, 2017 12:03 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Subject: Glyphosate AHS publication

Nancy—you may have already seen this but I just saw this published Thursday.

Glyphosate Use and Cancer Incidence in the Agricultural Health Study | JNCI: Journal of the National Cancer Institute | Oxford Academic:

https://academic.oup.com/jnci/article/doi/10.1093/jnci/djx233/4590280

Thanks,

Kaitlin

Sent from my iPhone

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Sun 11/5/2017 10:19:25 PM
Subject: RE: year-end BiOps

Nancy

Ex. 5 - Deliberative Process

If this were easy, it would have been resolved 20 years ago…

Mike

From: Beck, Nancy
Sent: Friday, November 3, 2017 6:09 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Fwd: year-end BiOps

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

Begin forwarded message:

From: "Dyner, Mark" <dyner.mark@epa.gov>
Date: November 3, 2017 at 5:03:09 PM EDT
To: "Keigwin, Richard" <Keigwin.Richard@epa.gov>, "Echeverria, Marietta" <Echeverria.Marietta@epa.gov>, "Beck, Nancy" <Beck.Nancy@epa.gov>, "Baptist, Erik"

<baptist.erik@epa.gov>
Cc: "Mclean, Kevin" <Mclean.Kevin@epa.gov>, "Perlis, Robert" <Perlis.Robert@epa.gov>, "Knorr, Michele" <knorr.michele@epa.gov>
Subject: year-end BiOps

Privileged/deliberative/attorney-client communication/do not disclose

Rick, Nancy, Erik:

Need to bring you all up to speed on NOAA's & DOJ's plans regarding the upcoming 12/31/17 BiOp deadline. Michele, Marietta & I just got off the phone with NOAA, FWS & DOJ staff. **Ex. 6 - Personal Privacy**

Ex. 5 - Attorney Client

Ex. 5 - Attorney Client

Happy to talk this through with you both if you'd like. Thanks.

Mark

Mark Dyner

Office of General Counsel

(202) 564-1754

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 11:10:17 PM
Subject: RE: EDSP

Nancy

Very nice note…

Mike

From: Beck, Nancy
Sent: Thursday, October 19, 2017 8:08 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: FW: EDSP

FYI, forgot to cc you on this one.

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Beck, Nancy
Sent: Thursday, October 19, 2017 8:05 AM
To: Barone, Stan <Barone.Stan@epa.gov>; Richard Keigwin (Keigwin.Richard@epa.gov) <Keigwin.Richard@epa.gov>

Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Louise Wise (Wise.Louise@epa.gov) <Wise.Louise@epa.gov>
Subject: EDSP

Stan and Rick,

Ex. 5 - Deliberative Process

If you both could assign someone to work on this and get back to us by Mid November, that would be ideal.

Please let me know if you have questions or concerns.

Thanks,
Nancy

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

To: Kaiser, Sven-Erik[Kaiser.Sven-Erik@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Hanley, Mary[Hanley.Mary@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Keller, Kaitlin[keller.kaitlin@epa.gov]; Jakob, Avivah[Jakob.Avivah@epa.gov]
Cc: Bolen, Derrick[bolen.derrick@epa.gov]
From: Dourson, Michael
Sent: Tue 10/24/2017 11:42:22 PM
Subject: RE: SEPW Minority Letter to Dr. Dourson
2017 10 24 Letter to Dourson_Adviser_with QFRs.pdf

Dear Colleagues

Here are some draft answers. Of course, please feel free to annotate them as needed. I would be more than happy to answer additional questions.

Cheers!

Michael

From: Kaiser, Sven-Erik
Sent: Tuesday, October 24, 2017 4:53 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Hanley, Mary <Hanley.Mary@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Keller, Kaitlin <keller.kaitlin@epa.gov>; Jakob, Avivah <Jakob.Avivah@epa.gov>
Cc: Dourson, Michael <dourson.michael@epa.gov>; Bolen, Derrick <bolen.derrick@epa.gov>
Subject: RE: SEPW Minority Letter to Dr. Dourson

OCSPP Team – thanks for handling. For reference, here's a similar exchange regarding Susan Bodine (incoming, response and attachments included). Please let me know if any questions. Best,

Sven

Sven-Erik Kaiser

U.S. EPA

Office of Congressional and Intergovernmental Relations

1200 Pennsylvania Ave., NW (1305A)

Washington, DC 20460

202-566-2753

From: Beck, Nancy
Sent: Tuesday, October 24, 2017 4:45 PM
To: Hanley, Mary <Hanley.Mary@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Keller, Kaitlin <keller.kaitlin@epa.gov>; Jakob, Avivah <Jakob.Avivah@epa.gov>
Cc: Dourson, Michael <dourson.michael@epa.gov>; Bolen, Derrick <bolen.derrick@epa.gov>; Kaiser, Sven-Erik <Kaiser.Sven-Erik@epa.gov>
Subject: FW: SEPW Minority Letter to Dr. Dourson

Mary,

Can you take the lead on getting a response drafted? We will likely need assistance from OCIR, OGC and OPPT.

Draft by next Friday? Is that possible?

Thanks.

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Kaiser, Sven-Erik
Sent: Tuesday, October 24, 2017 3:54 PM
To: Hanley, Mary <Hanley.Mary@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Jakob, Avivah <Jakob.Avivah@epa.gov>; Keller, Kaitlin <keller.kaitlin@epa.gov>
Subject: SEPW Minority Letter to Dr. Dourson

OCSPP Team – heads up on a letter to Dr. Dourson. I'm checking with OCIR management on handling and will let you know as soon as I hear something. Please let me know if any questions. Thanks,

Sven

Sven-Erik Kaiser

U.S. EPA

Office of Congressional and Intergovernmental Relations

1200 Pennsylvania Ave., NW (1305A)

Washington, DC 20460

202-566-2753

From: Lyons, Troy
Sent: Tuesday, October 24, 2017 1:14 PM
To: Aarons, Kyle <Aarons.Kyle@epa.gov>; Palich, Christian <palich.christian@epa.gov>; Kaiser, Sven-Erik <Kaiser.Sven-Erik@epa.gov>
Subject: FW: Letter to Dr. Michael Dourson

From: Ferrato, Margaret (Whitehouse) [mailto:Margaret_Ferrato@whitehouse.senate.gov]

Sent: Tuesday, October 24, 2017 1:04 PM
To: Lyons, Troy <lyons.troy@epa.gov>
Cc: Gaeta, Joe (Whitehouse) <Joe_Gaeta@whitehouse.senate.gov>; Leibman, Adena (Whitehouse) <Adena_Leibman@whitehouse.senate.gov>; Goldner, Aaron (Whitehouse) <Aaron_Goldner@whitehouse.senate.gov>
Subject: Letter to Dr. Michael Dourson

Hi Troy,

I hope you're well! Attached is a letter from members of the Environment and Public Works Committee to Dr. Dourson. Don't hesitate to reach out with any questions.

Best,
Maggie

United States Senate
WASHINGTON, DC 20510

October 24, 2017

Michael Dourson, Ph.D.
Adviser to the Administrator
Environmental Protection Agency
1200 Pennsylvania Avenue NW, 1101A
Washington, D.C. 20460

Dear Dr. Dourson:

It has come to our attention that you have recently been appointed to the position of "adviser to the administrator" at the Environmental Protection Agency (EPA) while your nomination to serve as EPA's Assistant Administrator of the Office of Chemical Safety and Pollution Prevention (OCSPP AA) is under consideration by the Senate. This appointment raises several concerns that we request you address before a Floor vote on your nomination, assuming the Environment and Public Works Committee agrees to advance it.

Your Appointment as Adviser to the Administrator

The Federal Vacancies Reform Act of 1998 provides, with limited exceptions, the "exclusive means for temporarily authorizing an acting official to perform the functions and duties of any office of an Executive agency … for which appointment is required to be made by the President, by and with the advice and consent of the Senate…." 5 U.S.C. § 3347. Further, as the Supreme Court held in *Buckley v. Valeo*, "any appointee exercising significant authority pursuant to the laws of the United States is an 'Officer of the United States,' and must, therefore, be appointed in the manner prescribed" in Article II, Section 2, clause 2 of the Constitution. 424 U.S. 1, 126 (1976). Accordingly, it would be unlawful for you to assume any of the delegated authorities of the OCSPP AA before the Senate confirms your nomination while serving as "adviser to the administrator."

Your appointment creates the appearance, and perhaps the effect, of circumventing the Senate's constitutional advice and consent responsibility for the position to which you have been nominated. Your improper involvement in EPA decisions could provide grounds for subjects of EPA regulations and oversight to challenge the legal validity of those decisions in court.[1] To ensure your appointment is not violating the Federal Vacancies Reform Act of 1998, please respond to the following:

- What is your official job title and type of appointment (e.g., non-career SES, Schedule C, administratively-determined)? Who, if anyone, are you supervising? What is your

[1] *See, e.g., National Labor Relations Board v. SW General*, 137 S. Ct. 929 (2017) (vacating an NLRB unfair labor practices complaint because the NLRB general counsel at the time had been appointed in violation of the Federal Vacancies Reform Act).

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relationship with the Acting OCSPP AA? If you have a written job description, please provide a copy.

- Has the Administrator formally delegated any duties of the OCSPP AA to you? Which, if any, OCSPP AA duties have you or are you presently performing?

- During your confirmation process, you entered into an ethics agreement that was approved by both EPA and the Office of Government Ethics and presented to the Senate Environment and Public Works Committee. Are you governed by the same ethics agreement in your current position? Please provide a copy of the signed Trump ethics pledge, and copies of any waivers to the pledge or recusal statements.

- You committed to notifying the Committee of all of your EPA email addresses "within seven days of using a new email address, including any aliases or pseudonyms." Please provide all email addresses you have used since starting at EPA and any new ones within seven days of their use.

- You also committed to "conducting all business using official email addresses or other means and to refrain from any mediums that are outside the Freedom of Information Act's reach." Do you commit to do the same pre-confirmation?

- During previous administrations, senior EPA managers' schedules have been available to the public on a daily basis. You also committed to "mak[ing your] calendar available on a timely basis" when asked if you would make your calendars available daily. Given your extensive work with industries regulated by EPA in the past, how do you define "timely," and if you are unwilling to commit to making your schedule available on a daily basis, why? Will you make your schedule available while in your current position? If so, how frequently?

- In your ethics letter to Kevin Minoli, EPA's designated agency ethics official, you stated upon confirmation you would resign form your positions with the University of Cincinnati, Toxicology Education Foundation, and Dourson, Dourson, and Fowler. Have you resigned from these positions upon accepting your current appointment as adviser to the administrator? If so, please provide copies of the written notification you committed to send Mr. Minoli upon terminating these positions. Have you, as promised in your ethics letter, refrained from "participat[ing] personally or substantially in any particular matter" involving these entities, or those with which you have a personal, financial, or professional interest, including North American Flame Retardant Alliance, Martha C. Dourson, LLC, and CreateSpace Independent Publishing Platform? Please also provide a list of all particular matters from which you have either been recused or for which you have requested waivers in order to continue your participation in.

Frank R. Lautenberg Chemical Safety for the 21st Century Act and Pollutants

You declined to answer several questions for the record from members of the Environment and Public Works Committee due to lack of familiarity with various issues or EPA's perspective on them as a nominee. We are particularly concerned about your incomplete answers to questions about the regulation of pollutants and chemicals, as well as implementation of the Frank R.

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Lautenberg Chemical Safety for the 21st Century Act, a broadly bipartisan bill that will be within your purview if confirmed. It has been widely reported that Nancy Beck, previously of the American Chemistry Council, has been working behind the scenes to undermine the protections Congress intended in this law.[2] Your prior association with the tobacco industry and your extensive work for the American Chemistry Council and other chemical manufacturers led *The New York Times* to deem you a "scientist for hire"[3] and accordingly raises similar concerns.

Now that you are "adviser to the administrator," we expect that you have familiarized yourself with these issues and can be more forthright in answering the questions we previously asked. For example:

- Of seven questions asked by Senator Carper related to specific chemicals and how EPA should protect people from exposures to chemicals when setting chemical safety standards, you provided only five partial responses. You did not provide all requested information in response to two questions submitted by Senator Carper that were related to funding sources and sponsors of work on specific chemicals that was performed by TERA. You also refused to answer any of Sena[illegible]Carper's eight questions related to implementation of the Toxic Substances Control Act.

- In response to three questions asked by Senator Whitehouse about EPA's role regulating mercury and mercury compounds under TSCA, you responded that you were unaware of the status of the agency's work. You declined to respond to Senator Whitehouse's question if you agreed with EPA's endangerment finding and instead indicated you are "not familiar with the details of EPA's endangerment finding and would need to do more research on the topic." You also declined to answer a question from Senator Whitehouse regarding how EPA should c[illegible]der the synergistic effects of chemicals when considering their approval under FIFRA.

- During repeated questioning by Senator Harris regarding your ethical and moral responsibility to recuse yourself from working on potential conflicts of interest, such as regulations pertaining to the chemical compound perchlorate, you repeatedly indicated that you would defer to the guidance of the EPA Ethics Office. In your responses, you declined to acknowledge that you possess the ability to proactively recuse yourself from such conflicts.

- In response to three questions asked by Senator Cardin about EPA's role regulating trichloroethylene, methylene chloride, and N-Methylpyrrolid[illegible] under TSCA, you responded that you were unaware of the status of the agency's work.

[2] Annie Snider and Alex Guillen, *EPA staffers, Trump Official Clashed over New Chemical Rules*, POLITICO, June 22, 2017, available online at: http://www.politico.com/story/2017/06/22/trump-epa-energy-chemicals-clash-239875.
[3] *Mr. Trump Outdoes Himself in Picking a Conflicted Regulator*, THE NEW YORK TIMES, Oct. 18, 2017, available online at: https://www.nytimes.com/2017/10/17/opinion/mr-trump-outdoes-himself-in-picking-a-conflicted-regulator.html.

ED_001803B_00003745-3

We request you provide more complete answers to the attached questions for the record on toxics and pollutants, informed by your current position at EPA. We look forward to your prompt responses as it will help inform how we engage with your nomination.

Sincerely,

Sheldon Whitehouse
United States Senator

Thomas R. Carper
United States Senator

Benjamin L. Cardin
United States Senator

Bernard Sanders
United States Senator

Jeffrey A. Merkley
United States Senator

Kirsten Gillibrand
United States Senator

Cory A. Booker
United States Senator

Edward J. Markey
United States Senator

Tammy Duckworth
United States Senator

Kamala D. Harris
United States Senator

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Inadequate Responses to QFRs from EPW Members

Senator Carper

Available online at: https://www.epw.senate.gov/public/_cache/files/f/0/f0729f1a-4385-453f-b7f8-442825a0721c/A681AA266D5CC024C98FCC85A944EB5E.senator-carper-questions-for-the-record-to-epa-nominees.pdf

Senator Whitehouse

1. Pursuant to the overhauled TSCA, EPA recently published its first inventory of mercury supply, use, and trade in the U.S., which have very little information because it did not benefit from the new reporting requirements. TSCA requires that EPA promulgate a mercury and mercury compound reporting rule by June 22, 2018 to assist in preparation of the inventory, the next one of which is required to be published by April 1, 2020.
a. Do you commit to completing the mercury and mercury compounds reporting rule by the June 22, 2018 deadline?

   I do not know the status of this rulemaking within the Agency. However, if confirmed I will work to make sure that the TSCA deadline for this rule can be met.

   b. Do you commit to identifying any manufacturing processes or products that intentionally add mercury or mercury compounds and recommend actions to achieve further reductions in such mercury use in the next inventory and publish that inventory by the April 1, 2020 deadline?

   As noted above, I do not know the status of these activities within the Agency. If confirmed, I will work to understand their status and to ensure that EPA is meeting the deadlines required by the Lautenberg amendments to TSCA.

2. Mercury was on the 2012 Workplan Chemical List, but was removed from the list in 2014 because EPA already knew how highly toxic mercury is, and the Agency indicated it would be undertaking activities to implement the Minamata Convention on Mercury anyway. Significantly, this action was taken well before the revised TSCA was enacted. Under the revised law, to facilitate meeting its Convention obligations to reduce mercury use in the production of switches and switches, the phase down of mercury use in polyurethane production, and to regulate mercury use in new products and processes, it may be necessary for EPA to identify mercury among the next round of chemicals prioritized for action under TSCA. Will you include mercury among the next round of chemicals prioritized for action under TSCA as needed to further reduce mercury use in products and processes, and meet our obligations under the Minamata Convention?

   I am not familiar with why mercury was removed from the 2014 workplan list. If confirmed, I will look into this and seek to ensure that EPA is taking necessary steps to further reduce mercury use in products and processes.

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3. How should the EPA consider the synergistic effects of chemicals when considering approval of these chemicals under FIFRA?

I am not familiar with how synergistic effects are evaluated currently in the pesticides program. If confirmed, I will seek to understand this to ensure that EPA's approach is appropriate.

4. In 2009, as mandated by the Supreme Court and backed by a robust scientific and technical review, the Environmental Protection Agency produced the Endangerment and Cause or Contribute Findings for Greenhouse Gases (GHGs) under Section 202(a) of the Clean Air Act. It found six greenhouse gases - carbon dioxide, methane, nitrous oxide, hydrofluorocarbons, perfluorocarbons, and sulfur hexafluoride - "taken in combination endanger both the public health and the public welfare of current and future generations." Do you agree with the EPA's endangerment finding? Why or why not?

I am not familiar with the details of EPA's endangerment finding and would need to do more research on the topic before answering this question.

Senator Markey

5. One of the most significant changes made to TSCA under the LCSA was the streamlined authority for EPA to require testing of chemicals by order. However, to our knowledge that authority has not yet been used in the 15 months since the law took effect.

   Given the importance of testing to fill data gaps, which is critical to both prioritization and risk evaluation -- and fundamental to a "risk-based" system, please tell us your plans for using the section 4 testing authority and approach for filling data gaps for both prioritization and risk evaluation."

   If confirmed, I will seek to better understand the Section 4 testing authority under TSCA. With this knowledge, I will work to ensure that it is appropriately used to help fill gaps for prioritization and risk evaluation.

6. The new law requires EPA to restrict new chemicals where the available data are insufficient to address their risks. How will you evaluate the adequacy of data in PMNs? What will you do to assure that new chemicals are adequately tested?

   I will use a weight of the evidence approach that considers all scientific evidence and information to evaluate PMNs.

7. The industry has pressured EPA to accelerate the completion of the review period for PMNs in order to reduce the PMN backlog. What steps will you take to assure that EPA does not sacrifice the rigor and thoroughness of the review process in return for speed?

   If confirmed, I will work closely with staff to completely understand the PMN review process to ensure its rigor and thoroughness.

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8. EPA staff has pointed to several ways industry can improve the efficiency of the review process by filing more robust PMNs that anticipate and respond to the likely concerns of EPA reviewers. What will you do to motivate industry to file more complete and accurate PMNs?

If confirmed, I will work closely with staff to completely understand the PMN process. It seems to me that if industry had a better understanding of the EPA evaluation approach, it should incentivize them to provide more complete and accurate PMN submissions.

Senator Duckworth

9. The Environmental Protection Agency (EPA) has said that exposure to cancer-causing chemicals in childhood can be as much as ten times as likely to lead to cancer than the same exposure to the same chemical in an adult. EPA has specific policies in place to account for these differences when it sets safety standards for chemicals.

   You have questioned these polices claiming in your papers that, "by about 6 months of age, children are usually not more sensitive to chemical toxicity than adults" and "we are not aware of reported cases of differential harm to infants or children from low levels of regulated chemicals, like pesticides or food additives." This research was funded by the American Chemistry Council and Croplife America.

   If you are confirmed, do you commit to apply, and not to weaken, EPA's current policies that account for the greater sensitivity and risk children may have from chemical exposures?

   If confirmed, I will apply EPA policies and guidance as they are appropriate and consistent with today's best available scientific evidence.

Senator Cardin

10. Before the end of the last Administration, EPA proposed to ban some uses of three dangerous chemicals using its new Toxic Substances Control Act authority. Trichloroethylene is a probable carcinogen that has been found in unsafe levels in household wells on Maryland's Eastern Shore. Accidental exposures to methylene chloride used in paint and furniture strippers has killed at least 56 people since 1980, including at least two Maryland residents. Exposure to a second chemical used in paint strippers, N-Methylpyrrolidone, is dangerous for pregnant women. If you are confirmed, do you commit to quickly finalize these rules and prohibit the uses of these chemicals?

    If confirmed I commit to quickly getting briefed on the status of these rules so that I can better understand them and the prohibitions proposed.

ED_001803B_00003745-7

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 11/22/2017 7:16:44 PM
Subject: RE: Senate Appropriations Chairman's Mark-up

Hmm… very interesting.

From: Beck, Nancy
Sent: Wednesday, November 22, 2017 8:49 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: FW: Senate Appropriations Chairman's Mark-up

FYI..

See chairmans mark summary

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Berkley, Bruce
Sent: Tuesday, November 21, 2017 12:28 PM
To: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Morales, Oscar <Morales.Oscar@epa.gov>; Keigwin, Richard <Keigwin.Richard@epa.gov>; Layne, Arnold <Layne.Arnold@epa.gov>; Hughes, Hayley <hughes.hayley@epa.gov>; Katz, Brian <Katz.Brian@epa.gov>; Calloway, Kennetta <Calloway.Kennetta@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Mottley, Tanya <Mottley.Tanya@epa.gov>; Hartman, Mark <Hartman.Mark@epa.gov>; Burns, Mike <Burns.Mike@epa.gov>; Richardson, Vickie <Richardson.Vickie@epa.gov>; Barber, Delores <barber.delores@epa.gov>; Barone, Stan <Barone.Stan@epa.gov>; Graves, Inza

<Graves.Inza@epa.gov>; Robinson, David <Robinson.David@epa.gov>; Scott, Gregory <Scott.Gregory@epa.gov>
Subject: Senate Appropriations Chairman's Mark-up

Hi everyone,

On November 20, 2017, the Senate Appropriations Committee released a Chairman's Mark showing its recommendations for the FY 2018 Interior, Environment and Related Agencies Appropriations Bill and Report. Attached is a summary by appropriations account. On the whole, OCSPP did well in the Senate Mark compared to both the House Mark and the President's Budget.

Greg has done some excellent analysis on the results of the Mark (see Excel spreadsheet). The Word file contains some of the key provisions. We have also included the House Mark language for your comparison.

Key Points:

-Provides $10M in TSCA in anticipation of collecting fees in 2018. This is similar to the $3M provided in 2017. Since we do not anticipate collecting TSCA fees in 2018, the SBO suggests having further discussions with OCFO recommending that this language not be included in the final Bill.

-Increases TSCA EPM by $5.6M over FY 17 Enacted. It appears that this increase is the result of a transfer of resources from ORD's IRIS program.

-Brings Pesticides program back to the minimum appropriation level.

If you have any questions, please let me know.

Thanks

Bruce Berkley

Deputy Director, OCSPP

Office of Program Management Operations

(202) 564-7802

To: Keigwin, Richard[Keigwin.Richard@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]
Cc: Keller, Kaitlin[keller.kaitlin@epa.gov]; Dinkins, Darlene[Dinkins.Darlene@epa.gov]; Sisco, Debby[Sisco.Debby@epa.gov]; Strauss, Linda[Strauss.Linda@epa.gov]; Dunton, Cheryl[Dunton.Cheryl@epa.gov]
From: Dourson, Michael
Sent: Wed 11/15/2017 1:17:41 PM
Subject: RE: Follow-up to 11/14 OPP General: Correcting Respirator Label Language

Rick

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Cheers!

Michael

From: Keigwin, Richard
Sent: Wednesday, November 15, 2017 7:22 AM
To: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>
Cc: Keller, Kaitlin <keller.kaitlin@epa.gov>; Dinkins, Darlene <Dinkins.Darlene@epa.gov>; Sisco, Debby <Sisco.Debby@epa.gov>; Strauss, Linda <Strauss.Linda@epa.gov>; Dunton, Cheryl <Dunton.Cheryl@epa.gov>
Subject: Follow-up to 11/14 OPP General: Correcting Respirator Label Language

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Attached is the briefing paper that we used during yesterday's discussion. I've also attached a draft of the updated section of the Label Review Manual, the resource that OPP risk managers use in their review of labels and a document that we've made available to stakeholders for many years, that would be the substantive document that would be issued for public comment.

Below is the current draft of the OPP Update that we would issue to open the comment period:

10/2/17: Draft OPP Update for Respirators

Revised Respirator Language for Draft Pesticide Label Review Manual

EPA is announcing the availability of revised respirator descriptions for pesticide labels

Ex. 5 - Deliberative Process

comment period will close on [DATE].

Ex. 5 - Deliberative Process

- Bring the respirator descriptions on pesticide labels into conformance with the current NIOSH respirator language;
- Ensure that pesticide handlers and their employers have the information they need to identify and buy the respirator required to provide needed protection;
- Delete outdated statements referring to respirators that no longer exist; and
- Clarify and update language to ensure the guidance is easy to comply with.

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Rick Keigwin

Director, Office of Pesticide Programs

US Environmental Protection Agency

To: Sands, Jeffrey[sands.jeffrey@epa.gov]
From: Dourson, Michael
Sent: Tue 11/14/2017 1:08:53 PM
Subject: RE: USDA

No, walking is great. See you at the East entrance at 1:35!

From: Sands, Jeffrey
Sent: Tuesday, November 14, 2017 8:07 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: USDA

If you don't mind walking, lets meet outside of East entrance at 1:35pm. Itll likely take us 15-20 to walk over to the South building.

From: Dourson, Michael
Sent: Tuesday, November 14, 2017 8:05 AM
To: Sands, Jeffrey <sands.jeffrey@epa.gov>
Subject: USDA

Jeff

I presume that you know where to go at USDA and how to get there. Please let me know if this is somehow different, and I will make arrangements.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

To: Zarba, Christopher[Zarba.Christopher@epa.gov]
From: Dourson, Michael
Sent: Tue 10/24/2017 10:41:43 PM
Subject: RE: Need help

Chris

I am free tomorrow at around noon and then after 4. But I will see you tomorrow at the 1:30 meeting. Perhaps we can squirrel away a bit of time after that meeting. On Thursday I am free before 9. I head back to Ohio on Thursday evening late, but have meetings all through day.

However, I am in all next week.

Cheers!

From: Zarba, Christopher
Sent: Tuesday, October 24, 2017 3:38 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: Need help

Sorry I don't. Will look to see if others do. Can we chat tomorrow? Just let me know what works.

Sent from my iPhone

On Oct 24, 2017, at 12:39 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

> Chris
>
> I promised one of the republican senators on the EPW committee the SAB notes on the WOTUS where I made the comment that the SAB committee had not considered the AWQC inpacts. Problem is I marked my copy of the notes and then the university

reclaimed my computer. Do you have my comments on this discussion, or perhaps just the notes from the meeting?

Cheers!

Michael

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 11/22/2017 7:16:26 PM
Subject: RE: Memo: Cross Agency Coordinating Committee on PFAS

Thanks Nancy…

From: Beck, Nancy
Sent: Wednesday, November 22, 2017 11:23 AM
To: Wise, Louise <Wise.Louise@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Mottley, Tanya <Mottley.Tanya@epa.gov>
Subject: Fwd: Memo: Cross Agency Coordinating Committee on PFAS

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

Begin forwarded message:

From: "Treimel, Ellen" <Treimel.Ellen@epa.gov>
To: "Flynn, Mike" <Flynn.Mike@epa.gov>, "Jackson, Ryan" <jackson.ryan@epa.gov>, "Bowman, Liz" <Bowman.Liz@epa.gov>, "Lyons, Troy" <lyons.troy@epa.gov>, "Dravis, Samantha" <dravis.samantha@epa.gov>, "Bennett, Tate" <Bennett.Tate@epa.gov>, "Bolen, Brittany" <bolen.brittany@epa.gov>, "Wooden-Aguilar, Helena" <Wooden-Aguilar.Helena@epa.gov>, "Wehrum, Bill" <Wehrum.Bill@epa.gov>, "Beck, Nancy" <Beck.Nancy@epa.gov>, "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>, "Breen, Barry" <Breen.Barry@epa.gov>, "Yamada, Richard (Yujiro)" <yamada.richard@epa.gov>, "Orme-Zavaleta, Jennifer" <Orme-Zavaleta.Jennifer@epa.gov>, "Shapiro, Mike" <Shapiro.Mike@epa.gov>, "Forsgren, Lee" <Forsgren.Lee@epa.gov>, "Best-Wong, Benita" <Best-Wong.Benita@epa.gov>, "Simon, Nigel" <Simon.Nigel@epa.gov>, "Starfield, Lawrence" <Starfield.Lawrence@epa.gov>, "Traylor, Patrick" <traylor.patrick@epa.gov>
Cc: "Grantham, Nancy" <Grantham.Nancy@epa.gov>, "Richardson, RobinH" <Richardson.RobinH@epa.gov>, "Hull, George" <Hull.George@epa.gov>, "Nickerson, William" <Nickerson.William@epa.gov>, "Owens, Nicole" <Owens.Nicole@epa.gov>, "Fonseca, Silvina" <Fonseca.Silvina@epa.gov>, "Hilosky, Nick" <Hilosky.Nick@epa.gov>, "Keller, Kaitlin" <keller.kaitlin@epa.gov>, "Plotkin, Viktoriya"

<Plotkin.Viktoriya@epa.gov>, "Peck, Gregory" <Peck.Gregory@epa.gov>, "Lewis, Josh" <Lewis.Josh@epa.gov>, "Miles, Erin" <Miles.Erin@epa.gov>, "Bloom, David" <Bloom.David@epa.gov>, "Elkins, Arthur" <Elkins.Arthur@epa.gov>
Subject: Memo: Cross Agency Coordinating Committee on PFAS

Good afternoon,

The Acting Deputy Administrator signed a memo today formalizing the PFAS Cross Agency Coordinating Committee (CACC). The CACC will work to identify the agency's priorities when it comes to addressing per- and polyfluoroalkyl substances (PFAS) and coordinate projects to address these priorities. The full memo is attached.

Please contact me with any questions. Thank you.

Ellen Treimel, Special Assistant

Office of the Administrator

U.S. Environmental Protection Agency

WJC-N 3310

202-564-0557 (w)

Ex. 6 - Personal Privacy (c)

To: Beck, Nancy[beck.nancy@epa.gov]; Keigwin, Richard[Keigwin.Richard@epa.gov]
From: Dourson, Michael
Sent: Wed 11/15/2017 1:07:24 PM
Subject: RE: Pre Brief Black Fly Registration Meeting with Congresswoman Cathy McMorris Rodgers

Nancy and Rick

Just a heads up to make sure you do not miss this event and are prepared for it. I have no information on it.

Cheers!

Michael

-----Original Appointment-----
From: Rodrick, Christian
Sent: Tuesday, November 14, 2017 3:27 PM
To: Rodrick, Christian; Beck, Nancy; Bodine, Susan; Wagner, Kenneth; Patrick, Monique; Schuster, Cindy; Holsman, Marianne; Lyons, Troy; Bolen, Derrick; Baptist, Erik; Ringel, Aaron; Traylor, Patrick; Shimmin, Kaitlyn; Jackson, Ryan; Willis, Sharnett; Keigwin, Richard; Kowalski, Ed; Kaiser, Sven-Erik
Cc: Dourson, Michael
Subject: FW: Pre Brief Black Fly Registration Meeting with Congresswoman Cathy McMorris Rodgers
When: Wednesday, November 15, 2017 12:30 PM-1:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where: DCRoomARN3428/OCIR

-----Original Appointment-----
From: Rodrick, Christian
Sent: Thursday, November 9, 2017 3:08 PM
To: Rodrick, Christian; Beck, Nancy; Bodine, Susan; Wagner, Kenneth; Patrick, Monique;

Schuster, Cindy; Holsman, Marianne; Lyons, Troy; Bolen, Derrick; Baptist, Erik; Ringel, Aaron; Traylor, Patrick; Shimmin, Kaitlyn; Jackson, Ryan; Willis, Sharnett; Keigwin, Richard; Kowalski, Ed; Kaiser, Sven-Erik
Subject: Pre Brief Black Fly Registration Meeting with Congresswoman Cathy McMorris Rodgers
When: Wednesday, November 15, 2017 12:30 PM-1:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where: DCRoomARN3428/OCIR

Pre-Brief Meeting in advance of Thursday, 11/16, meeting with Congresswoman Cathy McMorris Rodgers.

Call in info:

1 Ex. 6 - Personal Privacy

Ex. 6 - Personal Privacy

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Mon 11/20/2017 6:05:01 PM
Subject: RE: Monday's Section 5 meeting

I am at Ex. 6 - Personal Privacy all week.

From: Beck, Nancy
Sent: Monday, November 20, 2017 1:01 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: Monday's Section 5 meeting

What's the number we can call you.

_____
Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

On Nov 20, 2017, at 12:55 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

> Ok, give me a phone number if you need me for the 1 pm meeting…
>
> **From:** Beck, Nancy
> **Sent:** Monday, November 20, 2017 12:18 PM
> **To:** Dourson, Michael <dourson.michael@epa.gov>
> **Subject:** Re: Monday's Section 5 meeting
>
> Yes. Will call when I have a chance -- sadly may be after 6. But hopefully I will get a break before then.
>
> _____
> Nancy B. Beck, Ph.D., DABT
> Deputy Assistant Administrator, OCSPP
> P: 202-564-1273
> M: Ex. 6 - Personal Privacy

Beck.Nancy@epa.gov

On Nov 20, 2017, at 12:01 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

Nancy

Thanks! Most helpful. When you get a break, please give me a call later today at [redacted]
Ex. 6 - Personal Privacy

Cheers!

Mike

From: Beck, Nancy
Sent: Monday, November 20, 2017 11:34 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Fwd: Monday's Section 5 meeting

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

Begin forwarded message:

From: "Mclean, Kevin" <Mclean.Kevin@epa.gov>
Date: November 17, 2017 at 4:53:23 PM EST
To: "Beck, Nancy" <Beck.Nancy@epa.gov>, "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>, "Grant, Brian" <Grant.Brian@epa.gov>, "Baptist, Erik" <baptist.erik@epa.gov>, "Sadowsky, Don" <Sadowsky.Don@epa.gov>, "Wills, Jennifer" <Wills.Jennifer@epa.gov>, "Thaler, Elizabeth" <thaler.elizabeth@epa.gov>, "Morris, Jeff" <Morris.Jeff@epa.gov>, "Mottley, Tanya" <Mottley.Tanya@epa.gov>

Cc: "Hanley, Mary" <Hanley.Mary@epa.gov>, "Pierce, Alison" <Pierce.Alison@epa.gov>
Subject: Monday's Section 5 meeting

Attached is the options paper OGC has prepared for Monday's 11 am meeting.

To: Barone, Stan[Barone.Stan@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 11:07:16 PM
Subject: RE: Systematic Review Community of Practice - Oct 2017 meeting summary/announcements

Stan

Thanks!

Michael

From: Barone, Stan
Sent: Thursday, October 19, 2017 10:48 AM
To: Dourson, Michael <dourson.michael@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>
Subject: FW: Systematic Review Community of Practice - Oct 2017 meeting summary/announcements

Just to give some background on systematic review activities and coordination I provide the below as an FYI.

If you have any questions please let me know.

Stanley Barone Jr., M.S., Ph.D.

Acting Director Office of Science Coordination Policy (OSCP)

Office of Chemical Safety and Pollution Prevention (OCSPP)

US Environmental Protection Agency

202.564.1169 office

202.564.8452 fax

202.253.5079 mobile

From: Nichols, Jennifer
Sent: Thursday, October 19, 2017 10:08 AM
To: Lavoie, Emma <Lavoie.Emma@epa.gov>; Camacho, Iris <Camacho.Iris@epa.gov>; Henry, Tala <Henry.Tala@epa.gov>; Vogel, Dana <Vogel.Dana@epa.gov>; Lowit, Anna <Lowit.Anna@epa.gov>; Raffaele, Kathleen <raffaele.kathleen@epa.gov>; Thayer, Kris <thayer.kris@epa.gov>; Jones, Samantha <Jones.Samantha@epa.gov>; Schappelle, Seema <Schappelle.Seema@epa.gov>; Barone, Stan <Barone.Stan@epa.gov>; Reiley, Mary <Reiley.Mary@epa.gov>; Foster, Stiven <Foster.Stiven@epa.gov>; Hospital, Jocelyn <Hospital.Jocelyn@epa.gov>; Sams, Reeder <Sams.Reeder@epa.gov>; Wray, Austin <Wray.Austin@epa.gov>; Pope-Varsalona, Hannah <Pope-Varsalona.Hannah@epa.gov>; Benson, Amy <Benson.Amy@epa.gov>; Branch, Francesca <branch.francesca@epa.gov>; LaLone, Carlie <lalone.carlie@epa.gov>; Hoff, Dale <Hoff.Dale@epa.gov>; Dzubow, Rebecca <Dzubow.Rebecca@epa.gov>; Rimer, Kelly <Rimer.Kelly@epa.gov>; Axelrad, Daniel <Axelrad.Daniel@epa.gov>; Murphy, Deirdre <Murphy.Deirdre@epa.gov>; Radke-Farabaugh, Elizabeth <radke-farabaugh.elizabeth@epa.gov>; Arzuaga, Xabier <Arzuaga.Xabier@epa.gov>; Cogliano, Vincent <cogliano.vincent@epa.gov>; Gibbons, Catherine <Gibbons.Catherine@epa.gov>; Glenn, Barbara <Glenn.Barbara@epa.gov>; Hogan, Karen <Hogan.Karen@epa.gov>; Kraft, Andrew <Kraft.Andrew@epa.gov>; Luke, April <Luke.April@epa.gov>; Owens, Beth <Owens.Beth@epa.gov>; Pratt, Margaret <pratt.margaret@epa.gov>; Woodall, George <Woodall.George@epa.gov>; Congleton, Johanna <congleton.johanna@epa.gov>; Flowers, Lynn <Flowers.Lynn@epa.gov>; Cowden, John <Cowden.John@epa.gov>; Stanek, John <Stanek.John@epa.gov>; Lehmann, Geniece <Lehmann.Geniece@epa.gov>; Carlson, Laura <Carlson.Laura@epa.gov>; Reinhart, Paul <Reinhart.Paul@epa.gov>; Markey, Kristan <Markey.Kristan@epa.gov>; Kirk, Andrea <Kirk.Andrea@epa.gov>; Vasu, Amy <Vasu.Amy@epa.gov>; Hagerthey, Scot <Hagerthey.Scot@epa.gov>; Gatchett, Annette <Gatchett.Annette@epa.gov>; Dutton, Steven <Dutton.Steven@epa.gov>; Felker-Quinn, Emmi <felker-quinn.emmi@epa.gov>; Bennett, Micah <Bennett.Micah@epa.gov>; Schofield, Kate <Schofield.Kate@epa.gov>; Ridley, Caroline <Ridley.Caroline@epa.gov>; Suter, Glenn <suter.glenn@epa.gov>; Au, Sarah <au.sarah@epa.gov>; Walton, Barb <Walton.Barb@epa.gov>; Moya, Jacqueline <Moya.Jacqueline@epa.gov>; Euling, Susan <Euling.Susan@epa.gov>; Braverman, Carole <braverman.carole@epa.gov>; Wong, Eva <Wong.Eva@epa.gov>; Mottl, Nathan <Mottl.Nathan@epa.gov>; Phillips, Linda <Phillips.Linda@epa.gov>; Guiseppi-Elie, Annette <Guiseppi-Elie.Annette@epa.gov>; Tornero-Velez, Rogelio <Tornero-Velez.Rogelio@epa.gov>; McDow, Stephen <McDow.Stephen@epa.gov>; Thacker, Samuel

<Thacker.Samuel@epa.gov>; Fritz, Jason <Fritz.Jason@epa.gov>; Newcamp, Caitlin <Newcamp.Caitlin@epa.gov>; Bateson, Thomas <Bateson.Thomas@epa.gov>; Kopylev, Leonid <Kopylev.Leonid@epa.gov>; Rieth, Susan <Rieth.Susan@epa.gov>; Burden, Susan <Burden.Susan@epa.gov>; Yaquian-Luna, Jose <yaquian-luna.josea@epa.gov>; Lee, Sylvia <Lee.Sylvia@epa.gov>; Gallagher, Kathryn <Gallagher.Kathryn@epa.gov>; Thomas, Dana <Thomas.Dana@epa.gov>; Strong, Jamie <Strong.Jamie@epa.gov>; Fehrenbacher, Cathy <Fehrenbacher.Cathy@epa.gov>
Subject: Systematic Review Community of Practice - Oct 2017 meeting summary/announcements

SR Community – Below is the recap of our monthly meeting, held last week, and some additional announcements that may be of interest. Following from September's meeting on the evaluation of study quality, October's meeting was focused on automation of steps in the SR process. Summaries and links to the presentations are included below. Thanks to Alicia, Leonid, Ryan, and George for presenting!

Systematic Review Community of Practice | October Meeting Summary

Topic: Automation

•□□□□□□□□ **Kristan Markey (OCSPP)** – Kristan provided an overview of terms/approaches that are widely used in automation of the SR process, including rules-based algorithms, machine learning algorithms, and natural language processing. In addition, some pitfalls to automation were described. See slides here.

•□□□□□□□□ **Alicia Frame (OLEM) – Automation Approaches in SR** (slides here). Alicia provided three examples of automation in SR.

1. QC Prioritization of title/abstract screening: In this example, a training set of high and low priority reference as identified by manual review was uploaded in SWIFTReview, enabling the software to assign priority scores to references from the lit search results. This allowed the reviewer to examine the references ranked as high priority and determine if any were missed by manual screening.

2. QC broad search results: In this example, a narrow search was conducted to identify and

mark important references as included and mark non-relevant references as excluded in SWIFTReview. This parameterized model was then applied to results from a broader literature search to identify important references that were missed in the narrow search.

3. Topic modeling: In this example, a broad search was uploaded in SWIFTReview, which automatically builds topic models. Once topic models are created, they can be explored types of references within groups and groups and/or references can be selected or excluded for further screening.

• **Leonid Kopylev (NCEA) – Lesser known features of SWIFT** (slides here). Leonid described some retrospective analyses done on a lit search result (i.e., lit search results had been manually screened to identify important references) and demonstrated that SWIFT is good at identifying both 'good' and 'bad' references. For example, in one particular training set with 7 included and 7 excluded references, 30% of references with lowest ranking could be excluded and 95% of desired references would still be included. Leonid also described some considerations related to syntax in SWIFT, which is important to consider when creating search strings.

• **Ryan Jones (NCEA) -** Ryan discussed a lit search capability in HERO that allows a user to generate a "seed" or list of important references to be used in citation mapping, which automates human judgment and ranks references based on likelihood of relevance. This can be combined with results from traditional key word searches; the overlap between search methods results in 60% relevance. In addition, topic classification can be done with large lit search results to categorize studies as epidemiology, toxicology, ecology, or exposure.

• **George Woodall (NCEA)** – provided brief overview of pilot program to develop an agency-wide strategy for managing and communicating environmental health science information (slides here). Objective is to develop harmonized, integrated, and interoperable taxonomies and ontologies for specific knowledge domains. Some examples were provided.

Announcements & Activities

➢ Join the **Sharepoint** site! This is critical to managing our membership and communications starting in the Fall.

➢ **International Collaboration for the Automation of System Reviews (ICASR)** – meeting in London, October 17-18

➢ **Joint EFSA/EBTC scientific colloquium on evidence integration in risk assessment: the science of combining apples and oranges** – meeting in Lisbon, October 25-26

- BioCreAtIvE VI Challenge and Workshop – Bethesda, MD, October 18-20. The Critical Assessment of Information Extraction systems in Biology challenge evaluation consists of a community-wide effort for evaluating text mining and information extraction systems applied to the biological domain.

- Please let us know if you have any ideas, question, concerns, etc!

Next Meeting: November 14, 2017
Topic: TBD

-Jennifer Nichols, Emma Lavoie, Kristan Markey, Xabier Arzuaga, Emmi Felker-Quinn

---

Jennifer L. Nichols, Ph.D.

Toxicologist

National Center for Environmental Assessment

U.S. EPA | Office of Research and Development

(919) 541-0708

To: Fugh, Justina[Fugh.Justina@epa.gov]
From: Dourson, Michael
Sent: Mon 12/4/2017 1:20:47 PM
Subject: RE: SRA Annual Meeting Plenary Session Monday morning

Thanks!

From: Fugh, Justina
Sent: Saturday, December 2, 2017 4:23 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: SRA Annual Meeting Plenary Session Monday morning

Go, speak, eat and drink! You are participating in your EPA capacity, so you may partake of any event or meal that is provided to all participants on the day you are there. Enjoy!

Justina

Sent from my iPhone

On Dec 1, 2017, at 7:58 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

> Justina
>
> So I will be going to this annual Society for Risk Analysis meeting and likely invited to hospitalities where all folks are offered food. What is your call on this please?
>
> Cheers!
>
> Michael…
>
> … L. Dourson, PhD., DABT, FATS, FSRA
>
> Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Dourson, Michael
Sent: Tuesday, November 28, 2017 7:30 PM
To: Jackson, Ryan <jackson.ryan@epa.gov>
Cc: Lyons, Troy <lyons.troy@epa.gov>; Fugh, Justina <Fugh.Justina@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Beck, Nancy <beck.nancy@epa.gov>
Subject: FW: SRA Annual Meeting Plenary Session Monday morning

Ryan

I have a pending talk at the upcoming Society for Risk Analysis meeting in Crystal City, on December 11. The topic of the talk is shown in the emails below, but basically is me giving a few slides (5 at most) on risk analysis as an obsolete profession (or not). I am definitely in the "or not" camp. This commitment was made over 6 months ago.

At this point I am listed on the program as my EPA title below. Please advise if you need for me to change anything.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Pamela Williams [mailto:pwilliams@eriskscience s.com]
Sent: Monday, November 27, 2017 11:39 AM
To: 'Terje Aven' <terje.aven@uis.no>; Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: SRA Annual Meeting Plenary Session Monday morning

Agreed, I know Dr. Dourson has an excellent presentation related to risk analysis (or risk assessment) certification, so some discussion of this would be great.

From: Terje Aven [mailto:terje.aven@uis.no]
Sent: Monday, November 27, 2017 8:52 AM
To: Dourson, Michael
Cc: Pamela Williams
Subject: SV: SRA Annual Meeting Plenary Session Monday morning

Thanks a lot Michael, this is excellent, perhaps you can also think about what we should then do to meet this challenge. I know you would highlight training .. □

Best

Terje

Sendt fra E-post for Windows 10

Fra: Dourson, Michael <dourson.michael@epa.gov>
Sendt: Monday, November 27, 2017 3:56:12 PM
Til: Terje Aven
Kopi: Pamela Williams
Emne: RE: SRA Annual Meeting Plenary Session Monday morning

Terje

Thanks for the gentle reminder. I am of the mind to discuss the misunderstanding of our profession by unskilled folks, and the plethora of opinions, masquerading as erudite, flooding the market, so to speak. We are not obsolete, as much as we are emulated, unfortunately by folks who really do not understand the underlying science.

I will likely have a few slides as examples. I am thinking of a periodic table chart of chemical contaminants in various folks' bodies, and/or the blogs on various synthetic pesticides on our food, meanwhile ignoring, or more likely being ignorant of, the overwhelming proportion of pesticides in food that are naturally occurring.

I very much appreciate your efforts to pull this together and the initial slides from both you and Pamela.

Cheers!?

Michael

From: Terje Aven [mailto:terje.aven@uis.no]
Sent: Friday, November 24, 2017 4:58 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Pamela Williams <pwilliams@eriskscience s.com>
Subject: VS: SRA Annual Meeting Plenary Session Monday morning

Hi Michael,

How are things going concerning the preparation for the Panel ?

I know we are a little early, but we very much would appreciate some feedback before the end of the month to be able to plan the discussion in a good way.

Thanks a lot

Best

Terje

Fra: Terje Aven
Sendt: 20. oktober 2017 11:31
Til: doursoml@ucmail.uc.edu; ragnar.lofstedt@kcl.ac.uk; sguikema@umich.edu; kimt@aorm.com
Kopi: Pamela Williams <pwilliams@eriskscience s.com>
Emne: SRA Annual Meeting Plenary Session Monday morning

Hi all,

Thanks for participating in the panel **Risk Analysis: An Obsolete Profession?** It will be great :-)

I will have an introduction to the panel discussion, see enclosed preliminary slides with associated text (the last slides 16-25 are not planned to be presented).

After this introduction I give the word to Pamela, see her preliminary slides (not all of these will be used but they are included to make the presentation understandable).

The idea is that each of you has a prepared introduction of some 5-7 minutes, prepared with slides if you like, with clear statements –linked to abstract of the panel and hopefully inspired by mine and Pamela's slides.

We would not like to restrict creativity and what you find most important on this matter, so feel free to angle things in your way. Focusing on some few – one or two – themes is however recommended. To be able to lead the panel discussion in a good way, we think it is wise to have a process in advance – starting now – where we share some of the ideas we have. The aim of this dialogue is to make the panel as interesting as possible by being informed what is coming, so that one can get ideas for comments and questions. We would like to have a lively discussion so the point is not use this dialogue to obtain some unity or consensus at this stage (rather the opposite ☺)

Looking forward to hearing from you. What we ask from you now is an indication of what type of message – themes- that you would like to highlight - in text or using slides.

We would very much appreciate if we could get some input before 15 November.

Thanks a lot.

Enjoy the weekend.

Best

Terje

SRA Annual Meeting

Plenary sessions

Monday morning

Risk Analysis: An Obsolete Profession?

Risk analysis has advanced strongly the last 30-40 years. It is interdisciplinary in its scope but also developing as a science in itself. Yet we should ask, has it really evolved as it should? Is there a potential for reaching another level on both quality and outreach?

Is there a need for revitalization and new directions for the field and SRA, to strengthen the research and reflect current topics like resilience and security? Should we develop specific risk analysis certificates and educational programs?

The panel will discuss these topics - the role of risk analysis in society and how risk analysis as a field can be strengthened. We question, what does it really mean to be a risk analysis practitioner, professional and scientist?

Panel:

Chairs: Terje Aven and Pamela Williams

Michael Dourson, Seth Guikema, Ragnar Löfstedt, Kimberly Thompson

Terje Aven, University of Stavanger, Norway

Pamela Williams, E Risk Sciences

Michael Dourson, US Environmental Protection Agency (EPA) (waiting for final confirmation)

Seth Guikema, University of Michigan

Ragnar Löfstedt, Kings College, London

Kimberly Thompson, Kid Risk and University of Central Florida

To: Jackson, Ryan[jackson.ryan@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Morris, Jeff[Morris.Jeff@epa.gov]
From: Dourson, Michael
Sent: Wed 12/6/2017 2:03:27 PM
Subject: RE:

Ryan

I do not know this person and he did not show up in my society memberships. However, I will send my university colleagues a request. They have done some work with this group.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Jackson, Ryan
Sent: Wednesday, December 6, 2017 8:24 AM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>;

Morris, Jeff <Morris.Jeff@epa.gov>
Subject:

Do you guys know how to reach Ernie Rosenberg? He was formerly with the cleaning institute.

Ryan Jackson

Chief of Staff

U.S. Environmental Protection Agency

Ex. 6 - Personal Privacy

To: Palich, Christian[palich.christian@epa.gov]
Cc: Lyons, Troy[lyons.troy@epa.gov]
From: Dourson, Michael
Sent: Fri 12/1/2017 1:15:06 PM
Subject: RE: CRP: Dourson Meeting with Toomey Staff (1:00 PM)

Christian

Ok, I will be at your place around 12:20.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

-----Original Appointment-----
From: Lyons, Troy
Sent: Tuesday, November 28, 2017 7:03 PM
To: Lyons, Troy; Palich, Christian; Dourson, Michael

Subject: CRP: Dourson Meeting with Toomey Staff (1:00 PM)
When: Friday, December 1, 2017 1:00 PM-1:30 PM (UTC-05:00) Eastern Time (US & Canada).
Where: 248 Russell

WORKING CONTACT:
Tyler_Minnich@toomey.senate.gov
Tyler Minnich | Legislative Assistant

Office of U.S. Senator Pat Toomey

202-224-4254

To: Dourson, Michael[dourson.michael@epa.gov]
From: Dourson, Michael
Sent: Sun 11/5/2017 10:19:20 PM
Integrated Risk Information System.docx

Integrated Risk Information System (IRIS)

History

IRIS is a database containing information about a chemical's principle toxic effect and a concentration or dose at which the chemical will not likely cause this effect, even in sensitive humans. For chemicals where cancer is the principle toxic effect, this concentration or dose is associated with a very low risk of cancer (usually one chance in a million people). For chemicals that have another principle toxic effect (like liver toxicity), this concentration or dose is considered safe. Collectively, these concentrations or doses are referred to as **risk values**.

The determination of the principle toxic effect is referred to as **hazard identification** (although other effects at higher concentrations or doses are also described). The determination of these risk values is referred to as **dose response assessment**. These two processes, hazard identification and dose response assessment are part of **risk assessment** as described by EPA in many guidance documents based on the work of the National Academy of Sciences. Importantly, all EPA offices use risk values along with estimates of chemical exposure for rulemaking.

Up until 1995, IRIS contained risk values on over 500 chemicals and was considered to be the place where all important EPA risk values were placed. Two senior EPA technical groups met monthly to review all risk values before placing them on IRIS. Risk values on IRIS were considered to be THE EPA value for the particular chemical, and were to be used by all staff until more appropriate values were developed.

Ex. 5 - Deliberative Process

Political pressures

Ex. 5 - Deliberative Process

ED_001803B_00003783-1

One way forward

Time Frame

-
-
-
-
-

To: Sands, Jeffrey[sands.jeffrey@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Baptist, Erik[baptist.erik@epa.gov]; Bennett, Tate[Bennett.Tate@epa.gov]
From: Dourson, Michael
Sent: Sat 11/18/2017 1:32:12 AM
Subject: RE: Briefing Document
mdWPS-CT briefing document template.docx

Jeff

Very nice. Only one comment and it is likely a misremembering on my part. A famous quote by Hemmingway (I believe) is that if "I had more time, I would have written less." Seems to apply here.

Cheers!

Michael

From: Sands, Jeffrey
Sent: Friday, November 17, 2017 6:05 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Baptist, Erik <baptist.erik@epa.gov>; Bennett, Tate <Bennett.Tate@epa.gov>
Subject: Briefing Document

All,

Please review attached document over the weekend and please provide feedback for edits. This should summarize and reflect development conversations over the past couple days.

Additionally, it looks like our opportunity to catch up Monday AM prior to will not work. Lets try to connect over the weekend to work through any relevant issues, if possible.

Thanks for your consideration and have a nice weekend.

Jeffrey Sands

Senior Advisor to the Administrator for Agriculture Policy

1200 Pennsylvania Ave, NW

2415 WJC North

Washington, DC 20460

(202) 564-2263

To: Barone, Stan[Barone.Stan@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 10:36:57 PM
Subject: RE: Science integrity materials

Stan

Thanks for the upgrade in my position, but we will have to let the Senate confirm it! However, I very much appreciate receiving this information.

Cheers!

Michael

From: Barone, Stan
Sent: Thursday, October 19, 2017 11:12 AM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Subject: FW: Science integrity materials

The following was proposed for distribution last year and it has languished. We would like to get it out in the near future.

With arrival of new AA this may be a good time to send this out.

As I conveyed previously one of the major Science Integrity complaint/ grievance areas we have in the Agency and this AAship is around authorship and clearance issues.

In addition, as a related but longer term effort and part of public access transparency efforts we

have also been working with OSA to develop an electronic work flow for clearance and tracking that would provide ease of access for reporting of final products that have been cleared.

Stanley Barone Jr., M.S., Ph.D.

Acting Director Office of Science Coordination Policy (OSCP)

Office of Chemical Safety and Pollution Prevention (OCSPP)

US Environmental Protection Agency

202.564.1169 office

202.564.8452 fax

202.253.5079 mobile

From: Barone, Stan
Sent: Tuesday, September 12, 2017 2:29 PM
To: Beck, Nancy <beck.nancy@epa.gov>
Cc: Wise, Louise <Wise.Louise@epa.gov>; Morales, Oscar <Morales.Oscar@epa.gov>
Subject: FW: Science integrity materials

It would be great to send out an announcement of our OCSPP policy on clearance and authorship.

Included in this reminder is mention of SI policy and best practices.

Stanley Barone Jr., M.S., Ph.D.

Acting Director Office of Science Coordination Policy (OSCP)

Office of Chemical Safety and Pollution Prevention (OCSPP)

US Environmental Protection Agency

202.564.1169 office

202.564.8452 fax

202.253.5079 mobile

From: Barone, Stan
Sent: Monday, January 23, 2017 8:58 AM
To: Cleland-Hamnett, Wendy <Cleland-Hamnett.Wendy@epa.gov>
Cc: Wise, Louise <Wise.Louise@epa.gov>; Housenger, Jack <Housenger.Jack@epa.gov>; Keigwin, Richard <Keigwin.Richard@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Cunningham-HQ, Barbara <Cunningham-HQ.Barbara@epa.gov>; Morales, Oscar <Morales.Oscar@epa.gov>; Lowit, Anna <Lowit.Anna@epa.gov>
Subject: Science integrity materials

Attached are science integrity materials we discussed last year.

This includes all hands memo and I've attached 3 additional items for each office to modify and use in their Science Integrity discussions with staff.

•□□□□□□□□ OCSPP's clearance procedures for technical products (which includes clearance form, etc.),

•□□□□□□□□ the clearance tracker/spreadsheet, and

•□□□□□□□□ a presentation on best practices for scientific integrity.

These items were reviewed by EPA's SI official and I've incorporated her changes.

Stanley Barone Jr., M.S., Ph.D.

Acting Director Office of Science Coordination Policy (OSCP)

Office of Chemical Safety and Pollution Prevention (OCSPP)

US Environmental Protection Agency

202.564.1169 office

202.564.8452 fax

202.253.5079 mobile

To: Bahadori, Tina[Bahadori.Tina@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Morris, Jeff[Morris.Jeff@epa.gov]; Henry, Tala[Henry.Tala@epa.gov]; Hanley, Mary[Hanley.Mary@epa.gov]
Cc: Orme-Zavaleta, Jennifer[Orme-Zavaleta.Jennifer@epa.gov]; Rodan, Bruce[rodan.bruce@epa.gov]; Yamada, Richard (Yujiro)[yamada.richard@epa.gov]; Thayer, Kris[thayer.kris@epa.gov]; Lavoie, Emma[Lavoie.Emma@epa.gov]; Scheifele, Hans[Scheifele.Hans@epa.gov]
From: Dourson, Michael
Sent: Mon 12/4/2017 1:19:32 PM
Subject: RE: Slides we discussed

Tina

Thanks for this information. Very helpful. I believe that the OPP has a systematic way of training its younger staff to be better risk assessors. How does NCEA do this please?

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Bahadori, Tina
Sent: Sunday, December 3, 2017 11:23 AM
To: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Henry, Tala <Henry.Tala@epa.gov>; Hanley, Mary <Hanley.Mary@epa.gov>
Cc: Orme-Zavaleta, Jennifer <Orme-Zavaleta.Jennifer@epa.gov>; Rodan, Bruce <rodan.bruce@epa.gov>; Yamada, Richard (Yujiro) <yamada.richard@epa.gov>; Thayer, Kris <thayer.kris@epa.gov>; Lavoie, Emma <Lavoie.Emma@epa.gov>; Scheifele, Hans <Scheifele.Hans@epa.gov>
Subject: Slides we discussed

Dear OCSPP Colleagues,

Following up from our 'systematic review' discussions on Friday, I am forwarding the link to the slides we presented at the SAB's Chemical Assessment Advisory Committee (CAAC) meeting in September. We have also presented versions of these materials, in varying detail and depth to other audiences such as in NAS workshops, meeting with the European Food Safety Agency, meetings with state risk assessors, interagency meetings, and scientific conferences.

Link to slides:
https://yosemite.epa.gov/sab/sabproduct.nsf/AE79F54CBA716293852581A70074264A/$File/IRIS+Update.pdf

We look forward to our continued discussion.

Tina

==========

Tina Bahadori, Sc.D.

Director, National Center for Environmental Assessment (EPA/ORD/NCEA)

National Program Director, Human Health Risk Assessment (EPA/ORD/HHRA)

***New RRB Room 71210; Telephone: 202-564-7903; Mobile: 202-680-8771

To: Bolen, Derrick[bolen.derrick@epa.gov]
From: Dourson, Michael
Sent: Thur 11/16/2017 6:53:11 PM
Subject: RE: IRIS Paper
Integrated Risk Information System.docx

Derrick

Here you go!

Michael

From: Bolen, Derrick
Sent: Thursday, November 16, 2017 12:18 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: IRIS Paper

Mike-

Can you send me the two pager you sent to Richard Yamada?

Thank you,

Derrick Bolen

Integrated Risk Information System (IRIS)

History

IRIS is a database containing information about a chemical's principle toxic effect and a concentration or dose at which the chemical will not likely cause this effect, even in sensitive humans. For chemicals where cancer is the principle toxic effect, this concentration or dose is associated with a very low risk of cancer (usually one chance in a million people). For chemicals that have another principle toxic effect (like liver toxicity), this concentration or dose is considered safe. Collectively, these concentrations or doses are referred to as **risk values**.

The determination of the principle toxic effect is referred to as **hazard identification** (although other effects at higher concentrations or doses are also described). The determination of these risk values is referred to as **dose response assessment**. Importantly, all EPA offices use these risk values along with a particular chemical's **exposure assessment** for rulemaking. These three processes, hazard identification, dose response assessment and exposure assessment are used to characterize a chemical's potential risk to humans and are all a part of **risk assessment** as described by EPA in many guidance documents based on the work of the National Academy of Sciences. A similar risk assessment process is also used for protecting the ecosystem.

Up until 1995, IRIS contained risk values on over 500 chemicals and was considered to be the place where all important EPA risk values were placed. Two senior EPA technical groups met monthly to review all risk values before placing them on IRIS. Risk values on IRIS were considered to be THE EPA value for the particular chemical, and were to be used by all staff until more appropriate values were developed.

Ex. 5 - Deliberative Process

Political pressures

Ex. 5 - Deliberative Process

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One way forward

Time Frame

To: Dourson, Michael[dourson.michael@epa.gov]
From: Dourson, Michael
Sent: Sun 11/5/2017 10:19:18 PM
Subject: iris
Integrated Risk Information System.docx

Integrated Risk Information System (IRIS)

History

IRIS is a database containing information about a chemical's principle toxic effect and a concentration or dose at which the chemical will not likely cause this effect, even in sensitive humans. For chemicals where cancer is the principle toxic effect, this concentration or dose is associated with a very low risk of cancer (usually one chance in a million people). For chemicals where some other toxic effect is principle (like liver toxicity), this concentration or dose is considered safe. Collectively, these concentrations or doses are referred to as **risk values**.

The determination of the principle toxic effect is referred to as **hazard identification** (although other effects at higher concentrations or doses are also described). The determination of these risk values is referred to as **dose response assessment**. These two processes, hazard identification and dose response assessment are part of risk assessment as described by EPA in many guidance documents based on the work of the National Academy of Sciences. Importantly, all EPA offices use risk values along with estimates of chemical exposure for rulemaking.

Up until 1995, IRIS contained risk values on over 500 chemicals and was considered to be the place where all important EPA risk values were placed. Two senior EPA technical groups met monthly to review all risk values before placing them on IRIS. Risk values on IRIS were considered to be EPA values and to be used by all staff until more appropriate values were developed.

Ex. 5 - Deliberative Process

Political pressures

Ex. 5 - Deliberative Process

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One way forward

Ex. 5 - Deliberative Process

To: Washington, Valerie[Washington.Valerie@epa.gov]
From: Dourson, Michael
Sent: Thur 11/16/2017 6:50:39 PM
Subject: RE: Out today

Valerie

I talked with HR about my check. We are all covered.

Thanks!

Michael

-----Original Message-----
From: Washington, Valerie
Sent: Thursday, November 16, 2017 7:09 AM
To: Wooden-Aguilar, Helena <Wooden-Aguilar.Helena@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Greenwalt, Sarah <greenwalt.sarah@epa.gov>; Allen, Reginald <Allen.Reginald@epa.gov>
Subject: Out today

Gm All,

Ex. 6 - Personal Privacy

Sent from my iPhone

To: Jackson, Ryan[jackson.ryan@epa.gov]
From: Dourson, Michael
Sent: Tue 11/14/2017 1:23:33 AM
Subject: FW: PFOA
C-8_FINAL_CATT_REPORT_8-02.pdf

Ryan

Sorry, I hit the send button too quickly. Attached is the West Virginia report. Also of note is the text on page 9.

Cheers!

Michael

2.1 Pre Meeting Action Items

TERA is a nonprofit [501(c)(3)] corporation dedicated to the best use of toxicity data for the development of risk values. This organization is very well known and respected in the toxicology arena for their professionalism, wealth of knowledge, experience, and unbiased approach to deriving risk factors. All the non-TERA toxicologists on the CATT, whether from government agencies or industry, were in unanimous support of including TERA in this project.

From: Dourson, Michael
Sent: Monday, November 13, 2017 8:17 PM
To: Jackson, Ryan <jackson.ryan@epa.gov>
Subject: PFOA

Ryan

Here is the information you need for explaining the Dupont 1 ppb value (see red text below). It is from the West Virginia report in 2002. I would be more than happy to help you and Administrator Pruitt with any chemical toxicity question. I have studied most of the problematic

chemicals either while at EPA or afterwards, and sometimes both.

Cheers!

Michael

FINAL

AMMONIUM PERFLUOROOCTANOATE (C8) [PFOA]

ASSESSMENT OF TOXICITY TEAM (CATT) REPORT

August 2002

Department of Environmental Protection

State of West Virginia

3. 0 COMPARISON OF SCREENING LEVELS [SL] TO SITE-RELATED DATA

After the SLs for air, water, and soil were determined, DEP compared these SLs to the site-related data that has been collected to date. These comparisons are summarized below. The work of the CATT was only one facet of an investigation that continues beyond the issuance of this report. The GIST is expected to issue a report of the groundwater and surface water data in early 2003. The air modeling effort continues and is currently focusing on determining the results of the air emissions reduction efforts by DuPont required in the consent order as a 50% reduction in overall emissions (both air and water) by the end of 2003. Upgrades were completed in June 2002 which included the installation of a new scrubber and increased height of the primary C8

emissions stack.

-

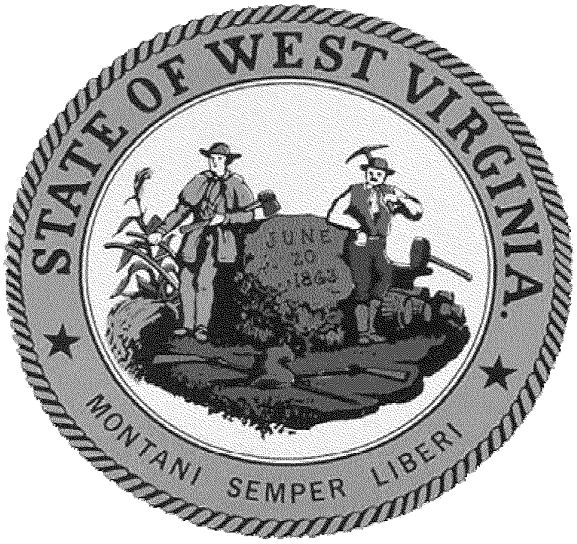
Water

To date, of the 188 samples collected from private wells, cisterns, and springs, 50 were used for drinking water and none exceeded the150 ppb health protective water SL for C8. Also to date, nine public water supply facilities in West Virginia have been analyzed for C8, including Belleville Locks and Dam, Blennerhassett Island, General Electric, Lubeck Public Service District (PSD), Mason County PSD, Parkersburg PSD, Racine Locks and Dam, New Haven Water Department, and Ravenswood. None of the drinking water from these facilities contained concentrations of C8 that exceeded the 150 ppb water SL. In fact, the concentrations of C8 in public water supplies were all below 2 ppb, below 15 ppb in private non-drinking water, and below 3 ppb in private drinking water wells in West Virginia. Samples were collected from Ohio public and private water supplies. Although C8 levels in some Ohio private water supplies were higher than those detected in West Virginia, none of these samples contained C8 concentrations above the water SL. These data have been provided to Ohio EPA and DEP will continue to share information with throughout the remainder of this investigation. **The DEP notes that the water SL [screening level] is higher than DuPont's internal community exposure guidelines for drinking water of 1 or 3 ppb; however, these guidelines were developed in the early 1990s and based solely on a two-week inhalation study from 1986. Since then significant additional toxicological data have been collected and the CATT water SL is based on a comprehensive examination of all available information.** Sampling of the Ohio River has begun; preliminary analytical results are expected from the laboratory in September 2002. To date, no analysis has been performed to measure C8 in soils in West Virginia on private property; therefore, no comparison can be made to the soil SL.

FINAL

AMMONIUM PERFLUOROOCTANOATE (C8)

ASSESSMENT OF TOXICITY TEAM (CATT) REPORT



August 2002

Department of Environmental Protection - ***promoting a healthy environment***

EXECUTIVE SUMMARY

Pursuant to a consent order signed November 14, 2001 between the West Virginia Environmental Protection and Health and Human Resources departments, and E. I. Du Pont de Nemours, Inc. (DuPont) the C8 (ammonium perfluorooctanoate) Assessment of Toxicity Team (CATT) was established to:

(1) determine risk-based human health protective screening levels (SLs) for this unregulated chemical in air, water, and soil;

(2) provide health risk information to the public; and

(3) determine an ecological health protective SL for C8 in surface water.

To date, two public meetings have been held in the vicinity of the DuPont Washington Works facility located near Parkersburg, West Virginia. Also, a team of 10 expert toxicologists have met and determined human health provisional risk factors for the oral and inhalation routes of exposure, and calculated health protective SLs based on these risk factors using Region 9 U.S. Environmental Protection Agency standard methodology. The results of the CATT's investigation are presented in summary below. The ecological SL for surface water currently is still in development. An addendum to this report is expected to be released in Fall 2002 presenting the surface water SL findings.

The methodology, overall process, and rationale utilized by the CATT to develop these risk factors and SLs are discussed, the members are listed, and a synopsis of the events leading to the consent order are presented herein. The intent of this report is to document the process and conclusions of the CATT in an effort to provide to the public a record of these activities. It is not intended to be a summary of all the toxicology information available on C8.

The risk factor or Reference Dose (RfD) for the oral route of exposure determined by the CATT for C8 was 0.004 milligrams per kilogram of body weight per day (mg/kg-day). A risk factor for the inhalation route of exposure or the Reference Concentration (RfC) of 1 micrograms per cubic meter of air ($\mu g/m^3$) was determined. The RfD or RfC is defined by EPA as an estimate (with uncertainty spanning perhaps an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a lifetime. Based on the oral RfD, health protective SLs were calculated for water of 150 parts per billion (ppb), and for soil of 240 parts per million (ppm). Based on the inhalation RfC, a health protective SL of 1 $\mu g/m^3$ was derived for air.

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ACKNOWLEDGEMENTS

The West Virginia Department of Environmental Protection wishes to thank the following agencies and organizations that joined us as primary participants in this investigation: West Virginia Department of Health and Human Resources; U.S. Environmental Protection Agency (EPA) Region 3, Office of Research and Development (ORD) and Headquarters; E. I. Du Pont de Nemours, Inc. (as well as their employees, consultants - Potesta & Assoc., Inc., laboratory – Exygen Research, Inc., and attorneys); Marshall University; Toxicology Excellence for Risk Assessment (*TERA*); and Menzie Cura & Assoc., Inc. Specifically, we thank the following EPA personnel for their technical support and camaraderie: Karen Johnson, Janet Sharke, Garth Connor, Roger Reinhart, and Mary Dominiak. We also thank the following organizations for their cooperation: EPA Region 5, Ohio EPA, and the National Institute for Chemical Studies.

We thank all the individual members of the C8 Assessment of Toxicity Team (CATT) for their participation and cooperation. In particular, we thank the following CATT members:

- James Becker, M.D., and Tracy Smith, M.S., of Marshall University for their professionalism, scientific knowledge, and common sense approach to communicating environmental health risks to the public.

- The toxicologists who embarked on an expedition to find the truth, the ambition of all noble scientists:

EPA

John Cicmanec, D.V.M., M.S., USEPA ORD
Samuel Rotenberg, Ph.D., USEPA Region 3
Jennifer Seed, Ph.D., USEPA Headquarters

TERA

Michael Dourson, Ph.D.
Joan Dollarhide, MS, MTSC, JD
Andrew Maier, Ph.D., CIH
Dan Briggs, Ph.D., DABT (note taker)

Agency for Toxic Disease Registry

John Wheeler, Ph.D.

DuPont

Gerald Kennedy
John Whysner, M.D., Ph.D., D.A.B.T. (consultant)

Invited guests:

John Butenhoff, Ph.D., 3M (study scientist)
Jim Sferra, MS, OEPA (observer)

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TABLE OF CONTENTS

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LIST OF TABLES

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1.0 INTRODUCTION

The investigation described herein was conducted pursuant to the November 14, 2001 Consent Order Number GWR-2001-019 between the West Virginia Departments of Environmental Protection (DEP) and Health and Human Resources (DHHR), and E. I. Du Pont de Nemours, Inc. (DuPont). A copy of this consent order is included as Attachment I. These actions were instigated by the presence of an unregulated chemical, ammonium perfluorooctanoate commonly called C8, in the Lubeck, W.Va. public water supply which is near the DuPont Washington Works (WW) facility in Washington, W.Va. A site map is included in Attachment IIc.

The consent order established two scientific teams: (1) the C8 Assessment of Toxicity Team (CATT), and (2) the Groundwater Investigation Steering Team (GIST). The CATT was tasked with investigating the toxicity of C8; developing provisional risk factors for the inhalation, dermal, and oral routes of exposure; and establishing human health protective screening levels (SLs) for air, water, and soil; investigating the ecological toxicity of C8 and determining an ecological health protective SL for surface water; and with communicating health risk information to the public. In the consent order DuPont agreed to meet these SLs at their WW facility, once developed, and that these SLs would remain in effect until superseded by U.S. Environmental Protection Agency (EPA) guidance. The CATT's activities and findings regarding the toxicity of C8, development of risk factors and SLs are presented in detail in Section 2 of this report. Slides presented at the two public meetings held thus far are provided in Attachment II. The investigation into the ecological toxicity of C8 and surface water SL development is scheduled for completion in Fall 2002. When finished, the surface water will be presented in an addendum to this report.

The GIST was established by the consent order to determine the extent and concentration of C8 in both groundwater and surface water. The activities of the GIST continue as of the issuance of this CATT report. The GIST will issue a report on the C8 analytical data for groundwater and surface water when that work is finished, scheduled for early 2003. Interim reports are available through the DEP Division of Water Resources (DWR). The groundwater investigation focused not only on the WW plant, but also on areas where C8 had been disposed, including the Local Landfill (on WW property), Dry Run Landfill (near the WW plant), and the Letart Landfill (30 miles south of the WW plant). Maps of the one-mile radius study area around these locations are included in the presentation of interim results at the second public meeting provided in Attachment IIc.

Summarized findings to date by the GIST are compared to the health protective water SL developed by the CATT in Section 3.0. Results of air dispersion modeling efforts thus far conducted by the DEP Division of Air Quality (DAQ) are compared to the air SL in Section 3.0 as well.

Background

The DuPont WW plant is located approximately 10 miles southwest of Parkersburg, W.Va. along state Route 61 in the rural hamlet of Washington, W.Va. This facility was established in the 1940s and currently is one of the largest DuPont enclaves in the world. DuPont has used C8 at this facility for more than 50 years as a surfactant in various manufacturing processes, including the production of Teflon. "C8" is the 3M trade name for its product that contains ammonium perfluorooctanoate (APFO) (CAS # 3825-26-1). In biologic media, APFO quickly dissociates to perfluorooctanoate, which is the anion of perfluorooctanoic acid (PFOA). The PFOA form has been identified as potentially toxic to animals. Throughout this report, C8 is used as terminology to include C8, APFO, or PFOA.

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The DEP became aware of and began investigating the presence of C8 in the Lubeck, W.Va. public water supply in November 2000. In Spring 2001, DEP received a letter requesting a formal agency investigation into DuPont's environmental releases of C8 and the presence of C8 in the Lubeck drinking water from attorneys representing a few citizens residing in proximity to the WW plant. The Lubeck public water supply well field lies approximately 3 miles south of the DuPont WW plant. Also around this time, DEP became aware that C8 was chemically similar to perfluorooctane sulfonate (PFOS), another perfluorocarbon manufactured by 3M, and that 3M had recently removed their Scotchguard product from the marketplace because it contained PFOS. From U.S. EPA Region 3 and Headquarters, DEP learned that 3M had undertaken a significant research effort into the toxicity of perfluorocarbons, particularly PFOS and including C8; that perfluorocarbons were potentially more toxic than previously thought; that 3M was submitting the new data to EPA under the Toxic Substances Control Act (TSCA); and that these data were publicly available under Administrative Record 226 (AR226). Additionally, DEP learned that DuPont was submitting toxicity data on C8 to EPA, as well.

DEP gathered data and met with DuPont and met with citizens attorneys in Spring 2001. The DEP, which regulates groundwater in West Virginia, was joined in the investigation by the DHHR, which regulates drinking water. The DHHR requested support from EPA Region 3 to enforce the National Safe Drinking Water Act. At the request of these agencies, DuPont supplied information regarding C8 and its use in manufacturing processes, its toxicity, and emissions. After several months of investigation and discussions, a consent order was signed in November 2001. A copy of the consent order is provided in Attachment I. It describes the tasks and members of the CATT and GIST. The DEP informed the public of the consent order and scheduled a public meeting to discuss the order.

The DEP held it's first public meeting regarding C8 on November 29, 2001 at Blennerhassett Junior High School which is located near the Lubeck and Washington communities. The meeting was spearheaded by the CATT and the GIST. The purpose of the meeting was to inform citizens of: (1) the requirements of the consent order; (2) the members and activities of the GIST; (3) their assistance was required to fill out and return a water use survey if they had groundwater wells, cisterns, or springs (particularly those used for drinking water), and to allow sampling of these water sources; (4) the members and activities of the CATT; (5) the available information regarding the toxicity of C8; and (6) the known current levels of C8 in the Lubeck public water supply, which were below 1 part per billion (ppb). At this meeting, James Becker, M.D. of Marshall University spoke regarding environmental exposures and risks in general, and Dee Ann Staats, Ph.D. (DEP) explained the CATT and GIST activities, the consent order, and known toxicity of C8. The slides from both presentations are provided in Attachment IIa.

By the end of January 2002, contractors were in place to assist the CATT and the GIST in their tasks. The GIST was headed by DEP and had members from DHHR, EPA, and Dupont. The CATT was headed by DEP and had members from DHHR, EPA, DuPont and the Agency for Toxic Substances Disease Registry (ATSDR). The DEP contracted with the National Institute of Chemical Studies (NICS), a nonprofit organization, which subcontracted the human and ecological toxicology work to the Toxicology for Excellence in Risk Assessment (*TERA*) group, also a nonprofit, which subcontracted the ecological toxicology work to Menzie Cura & Assoc., Inc. (MC). Both *TERA* and MC are well respected in the field of toxicology. The NICS subcontracted the risk communications tasks to Marshall University.

In March 2002, EPA Regions 3 and 5 signed a consent order with DuPont requiring the provision of alternative water to any resident in West Virginia or Ohio with C8 in drinking water at levels above 14

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ppb. The 14 ppb was an interim value in effect until the water SL was developed by the CATT. This value was taken from the final report by ENVIRON Int. Corp. (a consulting firm hired by DuPont) titled "A Hazard Narrative for Perfluorooctanoate (PFOA)", January 2002. An earlier draft, "A Review of the Toxicology of Perfluorooctanoate (PFOA)", November 2001, had proposed a drinking water value of 210 ppb. However, DEP's toxicologist, Dr. Staats, expressed concern over some of the assumptions made in the calculation of the 210 ppb to DHHR and EPA Region 3. The outcome of these discussions was a decision that a very conservative approach should be taken in the interim until the CATT water SL was developed. Therefore, 14 ppb was accepted as the interim water SL for alternative water provision. Note that this consent order was jointly signed by two regions of EPA because West Virginia is in Region 3 and Ohio is in Region 5. During the investigation, C8 had been found in the Little Hocking, Ohio public water supply. Also, note that DEP and DHHR invited Ohio EPA to join the CATT and GIST as observers, but not as members because this would have required renegotiating the consent order between West Virginia and DuPont.

TERA was assigned by DEP to review and compile the C8 toxicological information provided by DEP and to prepare for and hold a meeting of the CATT toxicologists during which the provisional risk factors and health protective SLs would be derived. The CATT toxicologists panel was comprised of 10 expert scientists with a collective span of experience of over 175 years and many specialties including endocrinology, veterinary medicine, cancer, and risk assessment.

TERA's efforts are described further in Section 2.1. By mid April 2002, *TERA* was prepared for the meeting. Also, *TERA* helped prepare the other toxicologists for the meeting by providing toxicity reports and summary information. The CATT toxicologists met on May 6 and 7, 2002 at EPA offices in Cincinnati, Ohio. The minutes of this meeting are provided in Section 2.2. The meeting lasted approximately 18 hours with roughly one-third of that time spent in discussions of C8's potential carcinogenicity. The oral provisional reference dose (pRfD) risk factor, and the two health protective SLs (for water and soil) based on this risk factor were developed at this meeting. The panel agreed that the toxicology database was insufficient to develop a dermal exposure pRfD. The inhalation provisional reference concentration (pRfC) risk factor and air SL developed at the meeting were only interim because additional data collection was necessary for their calculation. These data were collected and provided to *TERA*, who calculated the final pRfC and air SL, wrote a report describing this activity and forwarded it to the other CATT toxicologists for their approval. This document is provided in Section 2.3 as the post meeting action items. Both the meeting minutes and the post meeting action items were reviewed and approved by the panel of 10 highly qualified toxicologists.

An internal briefing for the DEP, DHHR, and EPA was held on May 8, 2002 to discuss the water and soil SLs. Rather than withhold this information while the meeting minutes report was prepared, DEP released the water and soil SLs so that the public would be informed of the status of their drinking water, and decisions could be made regarding the provision of alternative water supplies. In that spirit, DuPont and the public were informed – via a meeting with the above regulators and a press release, respectively - of the water and soil SLs on May 9, 2002.

A second public meeting was held at Blennerhassett Junior High School on May 15, 2002, to inform the public of the details of the SL development and of the groundwater C8 concentrations that had been detected at that point. Dr. Becker first spoke regarding environmental health risks in general. Dr. Staats described the process used by the CATT toxicologists to arrive at the water and soil SLs. Finally, David Watkins (DEP, GIST chairman) presented the C8 analytical data for private and public water sources. Slides of the presentations given at this meeting are provided in Attachment IIb.

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2.0 DEVELOPMENT OF RISK FACTORS AND SCREENING LEVELS

TERA was assigned to prepare for, host and document the meeting of the CATT toxicologists during which the provisional C8 risk factors (pRfDs and pRfC) would be developed by the group. The activities undertaken by *TERA* to prepare for the meeting are presented in Section 2.1. The actual minutes of the meeting are provided in Section 2.2., and the tasks conducted by *TERA* to develop the final air SL after the meeting at the direction of the panel are described in Section 2.3.

2.1 Pre Meeting Action Items

TERA is a nonprofit [501(c)(3)] corporation dedicated to the best use of toxicity data for the development of risk values. This organization is very well known and respected in the toxicology arena for their professionalism, wealth of knowledge, experience, and unbiased approach to deriving risk factors. All the non-*TERA* toxicologists on the CATT, whether from government agencies or industry, were in unanimous support of including *TERA* in this project.

TERA was tasked with compiling and reviewing the available toxicological data for C8. A literature search and review of these data was in draft by EPA Headquarters, this document was provided to *TERA*. The 3M submittals to AR-226 were provided to *TERA* by DEP. These data grew from a total of seven compact discs to 10 during the time period of this project. The AR-226 continues to grow with 3M submittals currently. The index of the first seven discs are provided in Attachment Va. Additionally, DEP conducted a literature search of C8 toxicity data on the National Library of Medicine's Medline and Toxline databases in June 2001. The results of these searches were provided to *TERA* by DEP as well. Also, documents submitted to DEP from DuPont in response to the EPA Region 3 request for information was made available to *TERA* by DEP, first by mailing relevant toxicology documents identified by Dr. Staats, and then by physically delivering all these documents to their Cincinnati office for *TERA* to sort and identify those deemed relevant and necessary for their work. Therefore, little literature searching or data retrieval was required of *TERA*.

After reviewing the existing C8 toxicology data, *TERA* selected studies that would be suitable for derivation of risk factors for the oral, dermal, and inhalation route of exposure. A list of the potential key studies was prepared. An indepth review of these studies was then conducted, and the details of the studies were summarized in tabular format. Next, *TERA* prepared a condensed table of these studies including critical effects and exposure levels identified by *TERA*, and blank columns for the other criteria necessary in the risk factor development process, such as the uncertainty factors. The documents listed below were provided to the other CATT toxicologists approximately two or three weeks prior to the meeting. *TERA* also prepared tables of suggested uncertainty factors, risk factors, and resulting SLs to DEP. These documents were discussed with Dr. Staats but were not distributed to the other toxicologists prior to the meeting in an effort not to influence their decisions, and not to give the false impression that the decisions on risk factor development had already been made and that the panel's purpose was simply to review *TERA*'s work. Rather, *TERA*'s suggestions would be presented at the meeting as a starting point for panel discussions and the development of the risk factors and SLs would be done as a group. The pre-meeting documents provided to the rest of the panel by *TERA* and DEP are contained in Attachment III. Also in Attachment III is a more detailed description of the decisions and methodology used by *TERA* in suggested risk factor development.

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2.2 CATT TOXICOLOGISTS MEETING MINUTES

Meeting of C8 Assessment of Toxicity Team (CATT) Toxicologists

May 6 and 7, 2002

Andrew W. Breidenbach Environmental Research Center, Cincinnati, Ohio

Attendees:

Voting Team Members

John Cicmanec, D.V.M., M.S., ACLAM, USEPA Office of Research and Development
Joan Dollarhide, M.S., M.T.S.C., J.D., Toxicology Excellence for Risk Assessment (*TERA*)
Michael Dourson, Ph.D., D.A.B.T., *TERA*
Gerald Kennedy, E. I. Du Pont de Nemours, Inc.
Andrew Maier, Ph.D., C.I.H., *TERA*
Samuel Rotenberg, Ph.D., USEPA Region 3
Jennifer Seed, Ph.D., USEPA Office of Pollution Prevention and Toxics (may abstain from voting)
Dee Ann Staats, Ph.D. (Chairperson), West Virginia Department Environmental Protection (DEP)
John Wheeler, Ph.D., D.A.B.T., Agency for Toxic Substances Disease Registry (ATSDR)
(representing West Virginia Department of Health and Human Resources [DHHR])
John Whysner, M.D., Ph.D., D.A.B.T. (consulting for DuPont)

Invited Guests

John Butenhoff, Ph.D., 3M Company (study director)
Jim Sferra, M.S., Ohio EPA (observer)

Note taker

Daniel Briggs, Ph.D., D.A.B.T., *TERA*

Introduction

The toxicologists on the C8 Assessment of Toxicity Team (CATT) met on May 6 and 7, 2002, to develop provisional reference doses (pRfDs) and screening levels (SLs) for ammonium perfluorooctanoate (C8) as specified in Consent Order GWR-2001-019 between the West Virginia Department of Environmental Protection, the West Virginia Department of Health and Human Resources, and E. I. Du Pont de Nemours & Co., (DuPont) dated November 14, 2001. These screening levels apply only to DuPont at their West Virginia facilities as specified in this consent order. Any use of these pRfDs or SLs for any other purpose or by any other regulatory agency is solely their choice and responsibility.

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The meeting opened with Dr. Staats announcing that this meeting was being held pursuant to the above-cited consent order as part of an enforcement action and was therefore closed to the public. Dr. Staats noted that, except for Dr. Butenhoff and Mr. Sferra who were invited guests, the panelists were named as part of the consent order and were free to enter into discussions and vote on issues. It was noted that Dr. Seed could abstain from voting at any time. The rules for the meeting were set forth as follows:

- The panel would strive for unanimous consensus, but if such consensus could not be reached, then the majority of votes would rule.
- The panel was expected to be cooperative and courteous with each other.
- The risk factors and screening levels would be developed together as a group, rather than simply by reviewing the work and suggestions of *TERA*.
- Votes would be taken at each decision point. After panel discussion on each point, a motion would be made on the floor. The chair would then repeat the motion and verbally poll each panel member individually. The chair would always vote last in order to not influence the voting.

TERA recorded the official minutes for the meeting. However, the chair recorded supplemental notes, which were provided *TERA* to assist in the preparation of the final Meeting Minutes Report. It was noted that specific discussion comments or votes would not be attributed to panel members (i.e., no names would be used) in the meeting report in order to facilitate full and open discussion among the team. It was also noted that *TERA* would distribute a draft meeting report to the CATT panel for their review and incorporate panel comments as appropriate. Each panel member would be asked to sign a statement agreeing that the meeting report is an accurate representation of the discussion and conclusions of the CATT Team. The original signatures will remain on file with the DEP.

The sequence of discussion on Monday, May 6 was oral noncancer assessment; dermal noncancer assessment and on Tuesday, May 7 was cancer assessment; inhalation noncancer assessment; oral screening level; and interim inhalation screening levels. (Note that Dr. Seed left the meeting at 2:30 pm on Tuesday, May 7, 2002; she was present and joined in all discussions through the cancer assessment.) However, for clarity, the meeting report is organized according to noncancer (oral, dermal, inhalation) assessment, cancer assessment, and screening levels. Below, under each heading is a brief description of *TERA*'s opening comments, followed by the panel discussion, and then the outcome of the panel discussion.

Noncancer Assessment: Review of the Oral Studies

Prior to the meeting, *TERA* evaluated the available human and animal health effects studies for C8. (A list of the documents and studies included in *TERA*'s prior review is provided in the Attachments). *TERA* evaluated the pool of available studies to identify the key studies that could be selected by the CATT panel as the basis for the pRfD. In narrowing the list of available studies, the available data were evaluated weighing considerations such as observed effect levels, study duration and quality, and applicability to human health. The judgments were made in a manner consistent with hazard identification and dose-response assessment practices used in current U.S. EPA risk assessments. Studies were generally given greater consideration as potential principal studies if they were at least of subchronic duration; identified NOAEL/LOAEL boundaries on the low end of the range provided by all the data; and had robust design (e.g., diverse array of endpoints, sufficient number of animals). From the total pool of available studies, *TERA* developed detailed summary tables for each of the key

studies having potential for being selected as the principal study for derivation of the pRfD. The resulting detailed summary table of key studies was provided to the panel members prior to the meeting to facilitate the selection of the principal study by the CATT panel and is attached. Therefore, discussion of the oral studies at the meeting focused on the tables presented in the attachment which identified those studies of sufficient duration, content, and quality to merit consideration as the bases for deriving a pRfD. The tables present *TERA*'s selection of critical effect levels, and highlight the study data for key parameters that showed treatment-related changes.

At the opening of the meeting, the panel discussed whether all adequate studies had been included and whether any potential key studies were missing. One panelist asked why the 90-day Rhesus monkey study (Goldenthal, 1978b) had not been included. *TERA* responded that the Rhesus study was not considered to be as useful as the cynomolgus monkey study (Thomford et al., 2001) because it had fewer animals per group, and suggested a higher NOAEL/LOAEL boundary; however, findings from the Rhesus study would be discussed together with the cynomolgus study as supporting data. The panel confirmed that, to the best of their knowledge, the table included all of the toxicity work that should be considered in selecting principal studies for deriving the pRfD for C8.

After agreeing that all of the potential critical studies had been identified, the panel then discussed the merits of each of the studies, and the appropriate No-Observed-Adverse-Effect-Levels (NOAELs), Lowest-Observed-Adverse-Effect-Levels (LOAELs), and lower bounds on the benchmark doses (BMDLs) for each study.

Human Studies (Olsen et al. 2000; Olsen et al. 1998; Gilliland and Mandel 1996; Gilliland and Mandel 1993; Ubel et al. 1980)

TERA initiated the discussion by providing a brief synopsis on the potential utility of the available human health effects studies for deriving the pRfD. Two cohort mortality studies were available: (1) Ubel et al. (1980) reviewed the records of 180 deceased 3M employees for a period of 30 years (1948-1978) and found no significant difference between observed and expected mortality rates; (2) Gilliland and Mandel (1993) found no increases in mortality rates from liver cancer or liver disease in 3,537 (2,788 males and 749 females) exposed 3M workers for 35 years (1947 – 1983). Note that since the CATT meeting, a new epidemiological study on almost 4,000 (80% male) 3M workers has been completed which found no increase incidence of cancer in C8 exposed workers. Several cross-sectional studies of 3M workers (111, 80, and 74 males in 1993, 1995, and 1997, respectively) were available. However, these studies were noted as being limited for use in deriving the pRfD, since workers were exposed to unknown amounts of C8 for varying time periods, and no clear signs of toxicity (such as elevated serum levels of liver enzymes were reported). The mixed findings regarding changes in hormone levels were noted. It was noted that many of these studies provided data on serum levels of C8 (or serum fluorine levels), which could serve as a measure of exposure. However, the current toxicokinetics data were not viewed as sufficiently developed to conduct a quantitative extrapolation from the reported serum levels to equivalent oral doses in humans. Based on this introduction, the panelists were asked to comment on the human data and its usefulness for deriving the pRfD.

Key Panel Discussion Points: Panelists noted that, although limited, the existing human data are consistent with the animal data when exposure levels are considered. Although weaknesses in the epidemiology data were noted, one panel member commented that the human data are useful for hazard identification purposes, and provide some level of comfort in conducting the assessment since they do not identify adverse effects in chronically exposed workers. It was noted that a few of the

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human subjects had C8 serum levels comparable to those observed in animal studies [20 parts per million (ppm) or greater]. Other panel members described gaps in the human studies. Regarding the absence of effects observed in the epidemiology studies, the panel noted that the small number of female subjects and uncertainties in exposure levels for workers prevents the existing data from being used to rule out human toxicity. For example, the very small numbers of women in the studies prevent drawing a conclusion regarding female reproductive effects. One panelist noted that the increased blood level of estradiol reported in some subjects is not clinically significant. In addition, no adjustments were made for body mass index (BMI) variations among subjects. Since BMI is known to affect estradiol levels and in this study BMI was the only parameter to correlate with hormone levels, it was noted that it is unlikely that C8 exposure was related to increased estradiol levels. The panel discussed Gilliland and Mandel (1986), which reported six prostate cancer deaths overall and four among exposed workers. One panel member commented on the update to this study (no study report was provided), which showed no indication of increased risk of prostate cancer. This follow up study demonstrated that only one of the four workers with prostrate cancer were determined to have been exposed when work history records and blood levels of C8 were examined.

It was suggested that it might be possible to correlate C8 serum concentrations with lack of observed toxicity to estimate a human NOAEL. However, it was noted that the lack of clear exposure levels in the human studies precluded this type of analysis. Although C8 half-life determinations were conducted in some of the human studies, this information cannot be used to determine exposure doses because some exposure to the subjects may still be occurring. However, it is clear that humans do not have the major sex-related half-life difference that exists in rats. It was noted that a physiologically-based pharmacokinetic (PBPK) model is being developed, which may be useful in estimating exposure concentrations from human serum C8 levels. However, a panel member familiar with the status of this current toxicokinetic modeling effort, noted that the data are not sufficiently developed to use for quantitative risk assessment purposes at this time.

Outcome: The panel agreed unanimously that the human studies were not adequate to be used for quantitative dose-response determinations. The human studies have many substantial data gaps, such as low numbers of subjects and unknown exposure concentrations. No LOAEL was established and the exposure uncertainty does not allow identification of a clear NOAEL. In final comments made during polling of the panel, one panel member agreed with the group, but noted that the data could be used to develop a bounding estimate. A second panel member added that some evidence suggests the endocrine system as a target for C8 effects, and therefore, the human data might support the animal toxicity studies.

Definition of Adverse Liver Effect

TERA noted that in all experimental animal studies liver effects occurred. For the purposes of conducting this assessment, *TERA* defined adverse liver effects as the presence of histopathology (moderate grade hypertrophy would be considered sufficient) in addition to statistically significant absolute or relative weight changes, or a liver weight change of 10% or greater. A doubling of serum levels of liver enzyme activity (e.g., alkaline phosphatase (ALP), aspartate aminotransferase (AST), or alanine aminotransferase (ALT)) would also indicate an adverse liver effect. These adverse effects are used by other health organizations as well. The panel unanimously agreed with this general definition of adverse for liver effects, but noted that individual studies could demonstrate a continuum of liver effects that could be considered biologically significant.

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Palazzolo et al. 1993

This is a 90-day study in male rats in which animals received C8 at doses of 0, 0.05, 0.47, 1.44, and 4.97 mg/kg-day in feed. The major finding in this study was increased liver weight with histopathological findings such as moderate hypertrophy. Panelists were asked to comment on the data from this study; on the selection of study adverse effect levels; and on the usefulness of this study as the basis for deriving a pRfD.

Key Panel Discussion Points: The possible role of peroxisome proliferation in the observed liver effects was discussed. The panel discussed uncertainty in the relevance of this mechanism to humans. One panelist stated that when considering the relevance of peroxisome proliferation, it is important to consider both qualitative and quantitative issues. This panelist suggested that peroxisome proliferation may potentially occur in humans because the cellular receptor that modulates this reaction in rodents has been found in humans, but that this mode of action should be considered to be only qualitatively relevant to humans because the receptor is far less expressed in humans, and humans have not been shown to manifest a peroxisome proliferation response. It was noted that USEPA has an on-going project to investigate the relevance to humans of rodent peroxisome proliferation effects, but at this time EPA has no official policy on the significance of peroxisome proliferation for humans. It was also noted that IARC has also considered the issue of peroxisome proliferation and concluded that this mode of action is not relevant to humans if it has not been demonstrated to occur in human cells or primates treated with the chemical in question. (Note that the panel discussed the role of peroxisome proliferation as a potential mode of action for tumor formation later in the meeting. The results of this discussion are documented in the section on Cancer Mode of Action)

Discussion occurred regarding the usefulness of relative versus absolute liver weight in determining adverse effect levels. One panelist stated that changes in both of these parameters are preferred before designating a dose as an adverse effect level. However, most panelists considered a change in relative liver weight to be sufficient to designate a dose level as an adverse effect level. It was noted that liver weights in dosed animals in this study were comparable to control values after an 8-week recovery period; however, the panel agreed that this recovery should not influence selection of the NOAEL and LOAEL values.

Outcome: The panel agreed unanimously that 1.44 mg/kg-day is the LOAEL for this study because at this level statistically-significant increases in relative liver weight and CoA oxidase activity occur. In addition, hepatocellular hypertrophy of minimal severity or greater is observed in 14 of 15 animals at this dose, and in 2 of 15 animals at grade 2 or higher. The panel recommended that benchmark dose modeling be performed for the data based on grade 2 or higher hepatocyte hypertrophy. This modeling was conducted during the course of the meeting, resulting in a BMDL estimate of 1.3 mg/kg-day. It was noted that this BMDL is essentially the same as the LOAEL found in this study. Most panelists believed 0.47 mg/kg-day is the NOAEL because at this dose there are no statistically significant changes in either absolute or relative liver weight and only a "minimal" severity of hepatocellular hypertrophy is reported at this dose. However, one panel member preferred to call this a "minimal LOAEL" rather than a NOAEL, noting that dose-related changes in critical liver parameters had been established at the lower dose levels and suggesting that these could be part of the continuum of effects that might be considered a minimal LOAEL.

Goldenthal 1978a

This is a 90-day study in male and female rats in which animals received C8 in their feed at doses of 0, 0.56, 1.72, 5.64, 17.9, or 63.5 mg/kg-day for males and 0, 0.74, 2.3, 7.7, 22.4, or 76.5 mg/kg-day for females. This study is limited by the small number of animals (5/sex) in each dose group. Therefore, this study was not considered to be a key study. However, it was presented for the panel's consideration and comments because it includes female as well as male animals and the data on relative liver weights allow a BMD to be calculated.

Key Panel Discussion Points: One panelist noted that a sex difference was observed in this study. Another mentioned that this study demonstrates the importance of internal dose (C8 serum level), as compared to the administered dose.

Outcome: The panel agreed with the proposed NOAEL, LOAEL, and BMDL as presented by *TERA*. However, the panel also agreed unanimously that the study was not adequate to serve as the basis for deriving a pRfD because of limitations in the study (e.g., the small number of animals).

York 2002

This is a two-generation reproduction study in which male and female rats received C8 doses of 0, 1, 3, 10, and 30 mg/kg-day by gavage in distilled water. Parental animals were exposed through cohabitation and gestation to weaning of F1 animals, approximately 6 weeks. F1 animals were exposed from weaning until weaning of the F2 generation. The primary findings were increased liver weight and liver pathology in P and F1 generation male animals; however, it was noted that histology was conducted only when gross effects had been observed, and therefore liver histopathology data were not available for the control and low-dose F1 generation males.

Key Panel Discussion Points: One panelist stated that this was study was of excellent quality because it was conducted according to OPPTS guidelines for 2-generation studies. Two panelists noted that the degree of F1 generation exposure to C8 while *in utero* and while nursing was uncertain and may not have occurred at all because of rapid elimination of C8 from the systemic circulation of the female rats after it was administered via gavage. Therefore, the lack of reproductive toxicity in this study may not be meaningful. Other panelists agreed, but stated that the fact of rapid clearance resulting in decreased fetal exposure may not be relevant for humans because women do not have the same active secretory mechanism for C8 that exists in the female rat. Another panelist noted that rodent placenta provides less of an anatomical barrier than exists in primates. Another panelist observed that studies with radiolabeled C8 demonstrated that C8 could cross the placental barrier in rats. One panelist wondered whether female rat pups at weaning have developed the active secretory mechanism for C8 that exists in the mature females. Another panelist recalled data showing that weanling female rats were able to clear C8 faster than males, but not as fast as mature females. One panelist recommended that delayed sexual maturation and increased frequency of estrous cycles be included in the adverse effects noted for females for this study. A panelist pointed out that this study indicated a critical difference in the toxicity of C8 versus the structurally similar perfluorocarbon PFOS; in that PFOS caused fetal death at birth in a similarly designed study, while in this study C8 administration was associated with only a slightly statistically significant increase in fetal death at the post-weaning timeframe.

Outcome: The panel concluded that the LOAEL for males is 1 mg/kg-day. The males showed statistically-significant increases in liver and kidney weights at 1 mg/kg-day. No histology was conducted on liver and kidney at this dose level because no gross lesions were seen. However, given

the substantial histopathology noted at the next higher dose level (3 mg/kg-day), the panel believed pathology does exist at the 1 mg/kg-day level; therefore this level meets the agreed-upon definition of an adverse effect. The panel concluded that the LOAEL for females is 30 mg/kg-day. The females showed several adverse effects at this dose level, including increased mortality and decreased body weight. No NOAEL was identified for males; the NOAEL for females is 10 mg/kg-day. All of these values apply to both the P and F1 generation animals. Two panel members reviewed the BMDL modeling results, and agreed with the selection of 0.42 mg/kg-day as the study BMDL.

Riker Laboratories 1983

This is a chronic, 2-year study in male and female rats in which animals received C8 in feed at doses of 0, 1.3, and 14 mg/kg-day for males and 0, 1.6, and 16 mg/kg-day for females. The primary findings in this study are liver effects in male rats. However, it was noted that this chronic study also reported non-hepatic effects (ovarian stromal hyperplasia and ataxia) in female rats. Although this effect was not found in the subchronic study that included females (Goldenthal, 1978), the small number of animals in that subchronic study (n=5) may have limited the power of the study to observe these effects.

Key Panel Discussion Points: One of the panelists identified some copying errors in the tables (incidences of mammary fibroadenomas, Leydig cell adenomas, and ALT activity in the control group) and these values were corrected prior to the panel discussion (the attached table presents the corrected values). The panel disagreed with the study author's conclusion stated in the study report that the testicular vascular mineralization was a "spontaneous change occurring in aging rats" and that the ovarian stromal tubular hyperplasia was "equivocally related" to C8 administration because it did not progress. The panel considered both these effects to be biologically significant and relevant for determining adverse effect levels. One panelist stated that ovarian stromal hyperplasia is not commonly found in rats and noted that in this study the incidence of ovarian stromal hyperplasia in the control animals is zero. The panel discussed the relevance of the ataxia observed in females, but did not reach any conclusions about its possible biological significance. One panelist noted that at the time this study was conducted, the term "hepatic megalocytosis" was synonymous with the term "hepatic hypertrophy" currently in use. It was noted that the BMDL of 0.73 mg/kg-day calculated based on liver effects in males is consistent with the NOAELs for liver effects observed in other rat studies. In the initial summary table from which the panel was working it was noted that no BMDL was estimated for ovarian stromal tubular hyperplasia, since an adequate fit to the data was not achieved. One reviewer suggested that a model fit might be possible using log-transformed data, since the study results showed a clear log-related response curve. This approach was applied during the meeting, and resulted in a best estimate of the BMDL of 1.6 mg/kg/day.

Outcome: The panel agreed unanimously to the proposed NOAEL of 1.3 mg/kg-day for males, with a corresponding LOAEL of 14 mg/kg-day based on the following adverse effects: increased liver weight, hepatic cystoid degeneration, increased ALT enzyme activity, and testicular vascular mineralization. The panel agreed that the LOAEL in females was 1.6 mg/kg-day based on a statistically significant increase in the incidence of ovarian stromal tubular hyperplasia, and that this study did not identify a NOAEL for females. The panel further agreed that the estimated BMDL from this study is 0.73 mg/kg-day based on liver effects in males as the benchmark response.

Thomford et al., 2001

This is a 26-week study in cynomolgus monkeys, in which animals received C8 at doses of 0, 3, 10, or 30/20 mg/kg-day by gastric intubation of gelatin capsule. Gastric capsule intubation was chosen as the method of C8 administration to avoid emesis, which had occurred in the earlier Rhesus monkey study (Goldenthal et al., 1978b). Even so, several animals had problems tolerating the highest C8 dosing; as a result, the high dose was either reduced or in some cases, discontinued. Afterwards, time-weighted average doses were used to approximate the C8 dose given to the high-dose group. One animal died in the high dose group; primary findings included clinical signs and altered liver weight. *TERA* presented that altered liver weight was not considered an adverse finding.

Key Panel Discussion Points: At least two panelists believed that the degree of absolute liver weight increase (30%) noted at the 3 mg/kg-day dose should be sufficient to identify this dose as the LOAEL. Other panelists responded that this weight increase resulted from mitochondrial proliferation, and therefore was an adaptive response, not an adverse effect. They also pointed out that, unlike laboratory rodents, cynomolgus monkeys routinely exhibit large genetic variations. As a result, large differences in organ weights among these animals is relatively common and a 30% difference between groups – especially small groups, as in this study – is not necessarily biologically meaningful. Some panelists attempted to compare this study with the study conducted in Rhesus monkeys in order to help define the LOAEL, but this was not possible due to the uncertainty of dosing caused by the emesis that occurred in the Rhesus study. One panelist asked if the dosing technique (gastric intubation of the drug contained in gelatin capsules) might have contributed to a large range of C8 blood levels because of differences in capsule disintegration rates. Another panelist responded that this was unlikely because, while the data sometimes demonstrated large inter-animal variations in blood levels, the intra-animal variation over several dose administrations was small. It was noted that C8 serum levels were essentially the same in the low and mid-dose groups: 74, 80, and 120 μg/mL at 3, 10, and 30/20 mg/kg-day, respectively. The panel concluded that the similarities in serum C8 levels may explain the very similar effects observed between the 3 and 10 mg/kg-day dose groups. One panelist noted that protein-binding saturation was similar between the monkey and human.

Outcome: The panel agreed that the LOAEL is best described as "from 3 to 10 mg/kg-day" based on 30% increased absolute liver weight, and that a NOAEL does not exist for this study. At all three dose levels, statistically significant increases in absolute and relative liver weights occurred, but without accompanying histopathology. No clinical or histopathological evidence of organ damage occurred at any of the three dose levels. Dose-related trends toward lower T3 and T4 levels were observed, but these failed to achieve statistical significance, even at the highest dose. The panel concluded that these data are insufficient to identify any single dose as a LOAEL or NOAEL. Since the serum C8 levels were essentially the same for both the 3 and 10 mg/kg-day doses, the panel believed that designating a range of 3 to 10 mg/kg-day for the LOAEL is the best way to describe the study results.

Noncancer Assessment: Oral Hazard and Dose-Response Characterization

(Note: Dr. Seed abstained from voting during this part of the meeting.)

Critical Study and Point-of-Departure

The summary of NOAELs, LOAELs, and BMDLs unanimously agreed to by the panel is presented in Table 1 below. The individual study adverse effect levels were discussed by the panel for the purpose of selecting a critical study and effect level for derivation of the pRfD.

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Key Panel Discussion Points: The primary target organ for C8 is the liver, and males are clearly more sensitive to this effect than female rats. One panelist observed that the liver effects in rats may be related to peroxisome proliferation, and therefore may not be quantitatively relevant for humans. For this reason, the liver effects in rats might not be an appropriate critical endpoint Another panelist responded that, because of this, it was important to note that the monkey and rat LOAELs are in the same range, and since the liver effects in monkeys may not be related to peroxisome proliferation, liver toxicity might also be a relevant endpoint for humans. The observation of ovarian effects in female rats at the same LOAEL as liver effects in males was noted as a second reason to consider the rodent studies as an appropriate basis for deriving the pRfD.

Table 1. Summary of NOAELs, LOAELs, BMDLs, and Critical Effects for Key and Supporting C8 Studies

	Species	Sex	NOAEL	LOAEL	BMDL	Critical Effect
Key Studies						
Palazzolo et al. (1993)	Rat	M	0.47	1.44	1.3	Liver
York et al. (2002)	Rat	M	None	1	0.42	Liver
Riker Laboratories (1983)	Rat	F	None	1.6	1.6	Ovary
		M	1.3	14	0.73	Liver
Thomford et al. (2001)	Monkey	M	None	3-10	None	Liver
Supporting Studies						
Goldenthal et al. (1987a)	Rat	M	0.56	1.72	0.44	Liver
Goldenthal et al. (1987b)	Monkey	M,F	3	10	Not done	Clinical signs

Some panelists favored choosing the monkey study as the critical study, due to the closer biological relationship with humans as opposed to rats. It was also noted that the observed increase in liver weight in monkeys may not be related to peroxisome proliferation and, therefore, may be more relevant for human health risk assessment. Other panelists disagreed, pointing to the uncertainties in dosing and effects, the small number of animals per dose group, and the unclear boundary between NOAEL and LOAEL values. Also, it was noted that the monkey study could not be considered the critical study because the 90-day, two-generation, and two-year rat studies all have LOAEL, NOAEL, and /or BMDLs below the LOAEL range identified in the monkey study, and therefore based on selection of the critical study with the lowest adequate NOAEL/LOAEL boundary would support the use of the rodent studies.

The panel considered whether it would be better to base the pRfD on a NOAEL or on a BMDL. Some panelists thought a NOAEL basis is a simpler concept and would be easier to explain to the public. Others responded that the BMDL captures more information from the entire study (e.g., reflects information from the full dose-response curve, and variability in the dose-response data) and therefore is the better choice as the basis for the quantitative dose-response assessment. Another panel member mentioned that a NOAEL is not a "no effect" level, rather it reflects the proportion of the responding population that can physically be observed in an experimental situation. Therefore, the size of the population is important. The panel agreed to not rule out using either a NOAEL or BMDL, but instead to focus on the quality of each study and the lowest critical effect level it provided.

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The panel noted the unusually good agreement of the NOAELs and LOAELs from all the studies. The lowest NOAEL observed in one of the potential key studies was 0.47 mg/kg-day, from the 90-day rat study by Palazzolo et al. (1993). The lowest LOAEL observed in a key study was 1 mg/kg-day from the rat two-generation study (York et al., 2002). This study did not test doses low enough to identify a NOAEL; however, the BMDL value estimated for this study, 0.42 mg/kg-day, was essentially the same as the observed NOAEL from the 90-day study. Therefore, the panel agreed that the BMDL was an appropriate NOAEL surrogate for the two-generation study. The ovarian stromal hyperplasia reported in the chronic rat study (Riker Laboratories, 1983), provided a higher LOAEL than the two-generation study, and the BMDL for this effect resulted in the same value as the LOAEL. This demonstrates that the liver endpoint is the critical effect, because it occurs at lower doses.

Outcome: Because of the consistency in NOAELs/LOAELs and critical effect in all the key studies, the panel concluded that all studies could be considered co-critical studies and that all provide important information for human risk assessment. However, the panel unanimously agreed that the NOAEL surrogate from the two-generation study, a BMDL of 0.42 mg/kg-day, should serve as the point-of-departure for the pRfD. This value was selected since it represented the lowest NOAEL or BMDL, and provided the added consideration of having evaluated reproductive and developmental effects.

Uncertainty Factors

If adequate human data are available, these data are used as the basis for noncancer risk factor development. Otherwise, animal study data are used, along with a series of professional judgments that are incorporated into the risk factor as "Uncertainty Factors" and account for an assessment of the relevance and scientific quality of the experimental studies. There are five different uncertainty factors commonly used to address issues of biological variability and uncertainty. Two factors (Interspecies and Intraspecies) are used to address variability or heterogeneity that exists between animals and humans, and within different human populations. Three factors (Subchronic, LOAEL, Database) are used to address lack of information. Typically, the maximum total uncertainty factor that EPA will apply is 3000. If all five areas of uncertainty/variability are present warranting a total UF of 10,000, then EPA generally concludes that the uncertainty is too great to develop an RfD. The panel discussed each area of variability or uncertainty separately. A short introduction to each area of uncertainty is provided below to aid the reader in evaluating the discussions of the panel.

Intraspecies Variability (UF_H): This factor accounts for the natural differences that occur between human subpopulations and for the fact that some individuals may be more sensitive than the average population. This factor is composed of two subfactors – one to account for toxicokinetic differences (how the body distributes and metabolizes the chemical) and one to account for toxicodynamic differences (how the body responds to the chemical). If no information is available on human variability, then a default value of 10 is used. If adequate information is available on one of the two subcomponents, then this information is used along with a default value of 3 for the remaining subfactor. If data are available to adequately describe human variability in both subfactors, then actual data may be used to replace default values. In addition, if a RfD is based on human data gathered in the known sensitive subpopulation, a value of 1 may be chosen for this factor.

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The panel discussed the lack of available data describing human variability. One panelist suggested a comparison of human C8 blood levels and values from the animal studies. The highest human serum C8 level reported was 111 ppm, but the average was approximately 5 ppm. No effects were noted in the human subject with the highest blood level. Thus, at least some people achieved serum C8 levels equivalent to those that resulted in adverse effects in animal studies.

As noted in the discussion of the human data above, the panel acknowledged gaps in the data on human variability and inability to define the most sensitive subpopulation, and therefore concluded that the default value of 10 was appropriate for this factor.

Interspecies Variability (UF_A): This factor accounts for the differences that occur between animals and humans and is also thought to be composed of subfactors for toxicokinetics and toxicodynamics. If no information is available on the quantitative differences between animals and humans, then a default value of 10 is used. If information is available on one of the two subcomponents, then this information is used along with a default value of 3 for the remaining subfactor. If data are available to adequately describe variability in both subfactors, then actual data may be used to replace default values. In addition, if a RfD is based on human data, then a value of 1 is appropriate for this factor.

One panelist mentioned that EPA has often used a UF_A value of 3 in other assessments when extrapolating monkey data to humans, because the kinetics and dynamics of monkeys are assumed to be similar to humans. This assumption is based on the fact that rhesus monkeys and macaques share a 92% genetic homology with humans and because monkey studies are able to detect a much broader range of clinical findings and more specific histopathology than rodents. In addition, studies on other chemicals in which a good database exists in rodents, monkeys and humans demonstrate that results in monkey studies parallel the human effects more closely than results in rodent studies.

Another panelist agreed and said the half-life of chemicals in monkeys was usually closer to humans than to rats. Other panelists responded that for C8, the half-life in monkeys is about 30 days; and this is much less than the C8 half-life in humans, which is estimated to be greater than one year. It was noted, however, that data on C8 half-life in humans is limited.

Because no data are available to warrant moving from the default, the panel unanimously agreed that a UF_A value of 10 is appropriate with either the rat or monkey toxicology studies.

Subchronic to Chronic Extrapolation (UF_S): Because the RfD protects for a lifetime exposure, this factor is applied when the database lacks information on the health effects of the chemical following a chronic exposure. Two issues are considered when making judgment on the use of this factor – are there data demonstrating that different health effects are expected following chronic exposure than subchronic exposure, and are there data demonstrating that the observed health effects progress in severity as exposure duration increases? If the database contains no information on chronic exposure, a default value of 10 is often applied, unless other data suggest a lack of progression with exposure duration. If the database contains adequate chronic bioassays, then a value of 1 is appropriate. If there are data addressing only one of the two issues, then a default of 3 may be applied.

It was noted that the database for C8 contains an adequate chronic rat study (Riker Laboratories, 1983). In addition, a second chronic study (Biegel et al., 2001) was available, although this study focused primarily on tumorigenic mechanisms in rats. In addition, for the purpose of evaluating uncertainty factors, the human occupational studies were considered by the panel to be informative on the response (or lack thereof) of humans following long-term exposure. The database demonstrates that liver

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toxicity was the more sensitive endpoint in both subchronic and chronic studies. In addition, the database clearly demonstrates that liver toxicity does not progress in severity following chronic exposure. This conclusion is supported by the observation that the subchronic studies identified lower NOAELs for liver toxicity than the chronic studies. One panelist noted that the liver effect in rat progresses to cancer. However the panel concluded that the cancer effect was due to the peroxisome proliferation mechanism (as discussed below in the discussion of the cancer risk assessment). Based on these considerations, the panel unanimously agreed that a UF_S value of 1 is appropriate for the rat studies.

The panel also discussed whether a different value for UF_S would be appropriate if the monkey study had been used as the critical or co-critical study. One panelist observed that there were no data in monkeys regarding the progression beyond 26 weeks; another responded that there was no reason to think the effects in monkeys would be any more progressive than those in rats. Another panelist suggested that the toxicity of C8 in humans does not appear to be progressive. However, the panel agreed that there was some inherent uncertainty in the monkey study to justify use of the value of 3 for UF_S if the monkey study were the critical study.

LOAEL to NOAEL Extrapolation (UF_L): Because the RfD is considered to be a subthreshold value that protects against any adverse health effects, this factor is applied when the database lacks information to identify a NOAEL. If the database does not identify a NOAEL, then a default of 10 is used for this factor. If a NOAEL is used, a value of 1 is appropriate. Often, if the database does not identify a NOAEL, but the adverse effects observed are of minimal severity, then a default of 3 will be considered appropriate for use of a "minimal LOAEL". [1]

Several of the studies considered as co-critical identified NOAELs; the lowest NOAEL is 0.47 mg/kg-day from the 90-day study. Also, the BMDL estimated for the two-generation study was essentially the same as the observed NOAEL from the 90-day study. These NOAELs and BMDLs were based on well-conducted studies and their use as a basis of the pRfD is consistent with standard practice. Therefore, the panel had confidence that the C8 database has identified the threshold for toxicity in rats, and it unanimously agreed a UF_L value of 1 is appropriate for the critical effect in the rat studies.

The panel also considered the value of UF_L that would be appropriate if the monkey study were to be used as the critical study. Because there is no clear NOAEL value, the panel agreed that a value of 1 was not appropriate. However, because the effects seen at the low dose were limited to mild increases in liver weight without accompanying changes in histopathology, or any other effect, the low dose was considered to be a minimal LOAEL. Therefore, the panel agreed that a UF_L of 3 would be appropriate if the monkey study were to be used as the critical study.

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[1] EPA is currently discussing the application of UF_L when using a BMDL. A BMDL value represents the lower limit on the dose that should cause 10% of the experimental animals to respond with the effect that is being modeled. Because animal studies typically cannot detect a response less than 10%, an experimentally derived NOAEL also represents the dose that causes 10% of the animals to respond. For this reason, EPA has historically considered a BMDL to be a NOAEL surrogate and selected a UF_L value of 1 when a BMDL is used. Although EPA does not have official guidance on this issue, recent discussions in the agency suggest that if the effect being modeled for the BMDL is adverse, then the BMDL should be considered as a LOAEL. Currently, BMDLs are being evaluated on a case-by-case basis, considering the nature of the effect being modeled and the relationship of the estimated BMDL to observed NOAELs.

Database (UF_D): The database for deriving a high confidence RfD includes two chronic bioassays by the appropriate route of exposure in different species, one two-generation reproductive toxicity study, and two developmental toxicity studies in different species. The minimal database required for deriving a RfD is a single subchronic bioassay, that includes a full histopathology examination. The database factor is used to account for the fact that a potential health effect may not be identified if the database is missing a particular type of study. This factor may also be used if the existing data indicate the potential for a heath effect that is not fully characterized by the standard bioassays, for example neurotoxicity or immunotoxicity. If the database is complete, a value of 1 is appropriate. If only the minimal database is available, then a default of 10 is used. A value of 3 may be used if the database is missing one or two key studies.

The panel agreed that the oral database for C8 is complete. For the purpose of evaluating uncertainty factors, the panel felt that the human occupational studies provided sufficient information on the effects of long-term exposure in humans to function as a chronic bioassay. In addition, the consistency between the monkey and rat subchronic studies provides confidence that non-rodent species respond similarly to rats and that liver is a sensitive target organ in all species. Furthermore, a developmental toxicology study indicated that such effects only occurred at high concentrations, and reproductive effects were monitored in the 2-generation reproductive study.

Therefore, the panel unanimously concluded that a UF_D value of 1 is appropriate with either the rat or monkey toxicology studies selected as the critical study.

Outcome: The summary of the panel's unanimous conclusions regarding individual and composite uncertainty factors is presented in Table 2 below. The composite uncertainty factor is obtained by multiplying the individual factors. (Note, that following EPA convention, an uncertainty factor of 3 actually represents the log of the halfway point between 1 and 10. Therefore multiplying half-log values of 3 results in a full log value of 10, rather than 9 as would be expected for numeric multiplication.)

Table 2. Panel Recommendations of UF Selection for Oral pRfD

Study	UF_H	UF_A	UF_L	UF_D	UF_S	Composite UF
All Rat	10	10	1	1	1	100
Monkey	10	10	3	1	3	1000

Oral Reference Dose (RfD)

The final value of the RfD is obtained by dividing the point-of-departure by the composite uncertainty factor. As discussed above, the point-of-departure selected by the panel is the BMDL of 0.42 mg/kg-day estimated from the rat two-generation study (York et al., 2002) and the composite factor is 100. Therefore, the resulting pRfD is 0.42 ÷ 100, or 0.0042 mg/kg-day. Because of the lack of precision inherent in the RfD, only one significant figure is appropriate; therefore, this value is rounded to 0.004 mg/kg-day.

For comparison purposes, the panel considered the pRfD values that would result from choosing alternative NOAELs or BMDLs as the point of departure. This analysis is presented in Table 3 below:

Table 3. Comparison of pRfDs Derived Using Different Studies

Study	UF	NOAEL	RfD	BMDL	RfD
Palazzolo et al. (1993)	100	0.47	0.005	0.72	0.007
Riker Laboratories (1983)	100	1.3	0.01	0.73	0.007
York et al. (2002)	100	---	---	0.42	0.004
Thomford et al. (2001)	1000	3-10 (LOAEL)	0.003-0.01	---	---

Based on this review table developed by the panel, the pRfDs that could be derived from the C8 oral database range from 0.003 to 0.01 – at most a factor of 3 separates the different potential pRfDs. Considering that the definition of the RfD states that the RfD incorporates uncertainty spanning an order of magnitude (a 10-fold variation), the panel noted that close agreement of the potential pRfD values provides added confidence in the derived pRfD of 0.004 mg/kg-day.

Noncancer Assessment: Review of the Dermal Studies

(Note: Dr. Seed abstained from voting during this part of the meeting)

The data on C8 by the dermal route of exposure are limited. Other than acute lethality, skin sensitization, and irritation studies, the dermal database consists of only a single 2-week study.

Kennedy et al. 1985

This is a two-week study in male rats in which animals had C8 applied to the skin for 6 hours/day, 5 days/week at doses of 0, 4.2, 42, and 420 mg/kg-day. Although this is a short-term study, it is the only candidate for possible use in determining a reference dose for the dermal route of administration. The primary effects observed were increased liver weight and liver pathology. A panelist noted that the study design prevented animals from ingesting the dermally-applied material. Although the amount of material inhaled was considered to be low, some inhalation almost certainly occurred in the dosed animal because the control animals had detectable C8 blood levels. It was also noted that the consistency of the material applied to the animals varied among the dose groups, depending on the concentration of C8 in the material matrix. In all instances the amount of material on the skin was considerably thicker than a monolayer, and therefore, the applied doses might not reflect accurately the absorbed doses of C8 in this study.

Key Panel Discussion Points: One panelist stated that this study could provide potentially useful information because systemic effects are observed at dose levels below those which cause portal of entry effects (skin irritation). The panel discussed whether it would be appropriate to extrapolate the results of this study to longer durations in order to derive a dermal pRfD. The panel concluded that such extrapolation would not be advisable because of the possibility of unpredictable longer-term dermal effects. One panelist asked if route-to-route extrapolation could be done from the oral studies

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to estimate a dermal NOAEL or LOAEL. Other panelists thought this would not be possible due to uncertainties in the C8 toxicokinetics by oral versus dermal exposure routes. For example, enterohepatic circulation is known to occur following oral exposure, but would not occur following dermal exposure. Therefore, the toxicokinetics of C8 is different between the two routes of exposure. Regardless of the route of entry, C8 is not metabolized. Furthermore, no data on the dermal absorption rate were identified. One panelist noted that if the findings from this study were used to determine a reference dose, the resulting value would be higher than the reference dose obtained from the oral studies. Therefore, using oral studies to set the reference dose would be adequately protective, of systemic exposure via the dermal route. Another panelist agreed, stating that no dermal reference dose should be identified at all, and that a specific reference dose for dermal exposure was not needed.

Outcome: The panel agreed unanimously that this study should not be used to determine a dermal pRfD because of uncertainties inherent in the study design as noted in the discussion.

Noncancer Assessment: Review of the Inhalation Studies

(Note: Dr. Seed was absent during this part of the meeting)

The data on C8 by the inhalation route of exposure are limited. Other than acute lethality studies, the inhalation database consists of a 2-week study and a developmental toxicity study.

Kennedy et al. 1986 and Staples et al. 1981

Two inhalation studies were discussed as potential candidates for deriving the pRfC. Kennedy et al. (1986) reported a two-week study in male rats in which animals were exposed head-only 6 hours/day, 5 days/week to C8 air concentrations of 0, 1, 7.6, or 84 mg/m^3. The primary effects observed in this study at the mid-concentration included increased absolute and relative liver weight, supported by clinical chemistry and histopathology findings. The high concentration resulted in severe toxicity, including mortality in one rat. Other findings at the high concentration group were increased lung and testes weight. A concentration-dependent increase in the incidence of nasal and ocular discharge was noted.

A second potential critical study for deriving the pRfC was a developmental toxicity study by Staples et al. (1981). Pregnant rats were exposed whole-body 6 hours/day on gestation days 6 to 15 to C8 air concentrations of 0, 0.14, 1.2, 9.9, and 21.0 mg/m^3.

The panel agreed the Kennedy two-week study provided the highest quality data for possible determination of critical effects and provided a slightly lower NOAEL/LOAEL boundary, even though both studies used similar air concentrations. In addition, the Kennedy et al. (1986) study evaluated a broader array of systemic endpoints, and included a histopathology examination.

In describing their initial review of the study, *TERA* noted that EPA's RfC methodology states that the air concentrations to which animals are exposed are to be converted to "Human Equivalent Concentrations ($Conc_{HEC}$)" by applying dosimetric adjustments (USEPA, 1994). Dosimetric adjustments account for the different structure and surface area of animal respiratory tracts compared with humans. Different dosimetric adjustments are applied depending on where effects are observed. For example, a different dosimetric adjustment will be applied for liver effects than will be applied for lung effects. *TERA* noted that the key piece of data needed to calculate the $Conc_{HEC}$ is a description of the particle size distribution (i.e., the mass median aerodynamic diameter and geometric standard

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deviation or GSD). Data available from the published study did not provide complete information about the mass median aerodynamic diameter for the low-concentration group, or GSD for any exposure group. In order to facilitate the discussion of the study, *TERA* presented human equivalent concentrations for liver (extrarespiratory) and lung (pulmonary) effects from this study assuming either a monodisperse particle size distribution or a polydisperse particle size distribution. These results were presented to the panel as shown in Table 4 below.

Table 4. Preliminary $Conc_{HEC}$ Calculations from Kennedy et al. (1986)

Study Concentration[a]	GSD = 1.3 (Monodisperse)		GSD = 3 (Polydisperse)	
	Liver	Lung	Liver	Lung
1.0	0.6	0.018	0.5	0.09
7.6	4.6	0.14	4.0	0.70
84	67.7	17.7	46.9	7.4

a. All values are presented in units of mg/m^3.

Key Panel Discussion Points: It was noted that the inhalation database does not meet the minimum database requirements for determining an RfC of one subchronic 90-day study that includes histopathology of the respiratory tract, but that the consent order required a pRfC in order to set air screening levels. One panelist stated that it was not appropriate to extrapolate from oral studies to derive a RfC because of the absence of data on toxicokinetics differences between these routes (e.g., effects of enterohepatic circulation, or absorption).

One panel member indicated that the data needed to calculate the $Conc_{HEC}$ (i.e., the mass median aerodynamic diameter [MMAD] and geometric standard deviation [GSD]), but not reported, in the published study could be made available to *TERA* after the meeting. The panel agreed that these data should be provided to *TERA*, for calculation of the appropriate $Conc_{HEC}$ following the meeting. The panel then discussed whether the lung or the liver was the critical organ, recognizing that the final designation of critical effect could not be made until the correct $Conc_{HEC}$ is calculated. *TERA* raised the question of whether the reported increases in the incidence of nasal and ocular discharge should be considered an adverse effect. It was noted that this effect is not uncommon for the exposure protocol that was used, and the effect was seen in all groups. It was further noted that C8 is not an irritant, and that no nasal histopathology was observed in exposed animals. In selecting critical study concentrations the panel discussed the lung effects at higher doses. One panel member suggested that at the high concentration the overt pulmonary toxicity was observed due to the large particle burden. Uncertainties in interpreting the lung effects were raised by the panel. One panelist noted that the studies were too short to determine what effect chronic exposure would have on the respiratory tract. Another suggested that existing human data associated with the human study reports discussed earlier (pulmonary function testing of workers, etc.) might be useful in determining NOAEL/LOAEL values. After this discussion, the panel considered the study concentration of 7.6 mg/m^3 to be the NOAEL for pulmonary effects, with the LOAEL of 84 mg/m^3.

The panel next discussed the liver effects. It was noted that the observed increases in liver weight were consistent with the effects observed in the oral studies. Another panel member noted the increased alkaline phosphatase (AP) values observed at the higher doses were not necessarily the result of the types of liver effects seen in the oral and dermal studies, since increased AP levels often reflect disorders of biliary flow. One panelist questioned the ability of the study to detect systemic effects given the short exposure period and the kinetics of the compound; however, another panelist replied that the half-life of C8 in rats is 5 to 7 days, and the study design would have allowed achievement of

steady-state concentrations in the blood. The panel considered the study concentration of 1.0 mg/m^3 as the NOAEL for liver effects. However, one reviewer suggested that if the liver effects are found to be the critical effect based on the $Conc_{HEC}$, then benchmark concentration modeling should be conducted before assigning a critical effect level.

The panel considered the appropriate uncertainty factors for a pRfC, noting that the final choice of an appropriate value for some areas of uncertainty may change depending on whether lung or liver effects are found to be critical. (Note to the reader: Essentially the same areas of uncertainty are considered in developing a RfC as for the RfD. For a full explanation of the purpose for each factor, see the earlier discussion.) For the same reasons as discussed for the pRfD, the panel unanimously agreed that a value of 10 was appropriate for UF_H. When considering interspecies extrapolation, it is generally considered that the dosimetric adjustments used to derive the $Conc_{HEC}$ account for the toxicokinetic differences between animals and humans. Therefore, the uncertainty factor only needs to address the toxicodynamic differences. Since there are no data regarding dynamic differences between rats and humans, the panel agreed that the default value of 3 was appropriate for UF_A. Since the Kennedy study identified a NOAEL, the panel unanimously agreed that a value of 1 was appropriate for UF_L.

The panel considered that two of the factors, UF_S and UF_D, were related to the decision of whether lung or liver is the critical effect. If liver effects are determined to be the critical effect, then at least one panelist felt that UF_S, could be addressed with an uncertainty factor of 1 because the oral studies provided enough information to be confident that the liver effects would not progress in severity following a chronic inhalation exposure. However, other panel members stated that there were insufficient data to assess whether liver would continue to be the critical effect or to provide information on how the respiratory tract would respond following longer-term inhalation exposures, and that a value greater than 1 for UF_S was needed. For the UF_S and liver as the critical organ, the panel votes were 1, 3, or 10 with the majority choosing 3. If liver effects are determined to be the critical effect, then panelists were split on the value of the uncertainty factor for UF_D, choosing values of either 3 or 10 with the majority of the panel choosing 3. No unanimous consensus was reached on these two factors; however, a clear majority vote was reached on uncertainty factors of 3 each for UF_S and UF_D in reference to liver as the target organ.

If lung effects are determined to be critical, the panel was divided almost equally on the appropriate value for UF_S with opinions covering the full range of options from 1 to 3 to 10. Note however, that six scientists voted for a factor less than 10 (either 1 or 3) and five scientists voted for a value greater than 1 (3 or 10). Similarly, the panel was divided on the appropriate value for UF_D; panel opinions covered the full range of options from 1 to 3 to 10 with the majority of panelists choosing 3.

As noted above, after each discussion votes were taken on individual factors. These votes are shown in Table 5. Note that one scientist was reviewing the dosimetric adjustment calculations during this discussion and so was unable to vote on these UFs; also note that one more vote at any point in Table 5 would not have changed the final outcome. In addition, the panel did not reach consensus on the confidence in the RfC, with opinions ranging from "none" to "high" with the average being medium-to-low.

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Table 5. Tally of Panel Votes for UF_S and UF_D						
	UF_S			UF_D		
Factor	1	3	10	1	3	10
Liver as critical	1	6	1	0	6	2
Lung as critical	3	3	2	1	5	2

Outcome: One panelist reminded the group that the purpose of Kennedy et al., (1986) was to identify the inhalation hazard, not to look closely at NOAEL, LOAEL, etc. A prospective inhalation study designed to look more closely at the NOAEL/LOAEL aspects, to evaluate lesions as a function of exposure time, and to evaluate tissues of the respiratory tract using up-to-date methodology would be valuable and would allow a more focused evaluation of the RfC. Nonetheless, the panel agreed that a pRfC could be developed, but this agreement was not unanimous. The panel also recommended that *TERA* obtain additional data on the particle size GSD value to determine the $Conc_{HEC}$ corresponding to the NOAEL before determining whether the pulmonary or the hepatic effects are considered critical. If the liver effects are determined to be the critical effect, then BMD modeling should be done. The composite uncertainty factor was expressed as a range of 30 to 3,000. The final pRfC is presented in the Post Meeting Action Items.

Cancer Assessment

(Note: Dr. Seed abstained from voting during this part of the meeting)

U.S. EPA's 1999 Guidelines for Carcinogen Risk Assessment were used to frame the discussion of C8 carcinogenic potential. *TERA* opened the discussion with a short introduction to these guidelines, highlighting the recent focus on evaluation of the mode of action data in developing a weight of evidence characterization, and in deciding the most appropriate dose-response approach, linear or margin of exposure (MOE). It was noted that the EPA's 1999 guidelines would be used as the basis for the deliberations of the panel.

Cancer Hazard Identification and Mode of Action

The panel discussed the evidence for C8 carcinogenicity in humans and agreed that the human carcinogenicity evidence is inconclusive. Although four prostate tumors were reported in retired workers, three of these four cases now are known to have had minimal or no C8 exposure. (See Human Studies section for more detailed discussion.)

The panel noted that two animal carcinogenicity studies had been conducted. The first study (Riker Laboratories, 1983) reported treatment-related increases in Leydig cell adenomas and mammary gland fibroadenomas. The second study (Biegel et al., 2001) reported treatment-related increases in tumors in the liver, Leydig cells, and pancreas. Panelists noted that the tumors identified in the Biegel et al. (2001) study correspond to the triad of tumors associated with some chemicals that cause peroxisome proliferation. Other panelists agreed and suggested that a further examination of the data may indicate that this triad of tumors can be best addressed using a MOE approach. The panel also noted that the mammary fibroadenomas may require the default linear model because, following U.S. EPA cancer guidelines, no actual mode of action data for C8 and this tumor type are available to warrant moving from the default assumption. Each of the four types of tumors found in the two C8 animal

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carcinogenicity studies was then discussed in detail with regard to the weight of the evidence for the mode of action, and the evidence supporting a linear or MOE dose-response assessment approach. Listed below are the outcomes and discussions for each tumor type.

Liver tumors

Key Panel Discussion Points: The discussion on liver tumors focused on the role of peroxisome proliferation as the mode of action for the observed liver tumors. In relating this liver tumor effect to humans, one panelist said humans are much less sensitive to peroxisome proliferation than rats. Another panelist noted that IARC's approach for clofibrate and other non-genotoxic peroxisome proliferation chemicals was to assume that the mode of action was not relevant to humans if no evidence of peroxisome proliferation was observed in humans. Another panelist said that although rats may be more sensitive than humans from a toxicodynamic standpoint (due to interspecies differences in receptors), humans may be more sensitive from a toxicokinetic standpoint, since they clear C8 more slowly than rats. As a result, the panel member suggested that these two considerations would tend to decrease overall differences in species sensitivity. On the other hand, a panel member noted that no increased incidence of tumors have been found in people taking clofibrate, a known peroxisome proliferator, which suggests that humans are much less sensitive to peroxisome proliferation than rats and they may have no response at all. Based on these data, the panel member suggested that the lack of tumor development in humans exposed to C8 should not be discounted. The panel discussed differences in results between the two cancer studies. One panelist noted the studies have differences in their internal delivered doses because of differences in the animal diets. This could explain the difference noted in toxic effects.

Outcome: The majority of the panel agreed that the data indicate peroxisome proliferation is the mode of action for the liver tumors, and that although the liver tumor response is not likely to be quantitatively similar between rats and humans, the use of the liver tumor response data for human health risk assessment cannot be totally discounted. However, other scientists indicated that based on the lack of peroxisome proliferation in the non-human primate studies, the rodent liver tumors are not relevant at all to humans.

Leydig Cell Tumors

Key Panel Discussion Points: In reviewing the summary tables prepared for the meeting, one panelist noted that Leydig cell hyperplasia should be evaluated. In response, the hyperplasia data from Biegel et al. (2001) was reviewed by the panel. The panel developed Table 6 to facilitate the comparison on hyperplasia and tumorigenic outcomes.

Table 6. Summary of Beigel et al., 2001 Leydig Cell Data

	Pair fed controls	300 ppm
Liver carcinomas/adenomas	3/79	10/76*
Leydig ademonas	2/78	8/76*
Pancreatic carcinomas/adenomas	1/79	8/76*
Leydig cell hyperplasia	26/78	35/76

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The panel noted that no significant increase in Leydig cell hyperplasia was apparent from these data; however, due to different survival times between the two groups (C8 treated animals survived longer) a false positive effect could have occurred because older animals would have more time to develop naturally occurring tumors. It was noted that a more formal analysis would be needed to determine whether the incidence of Leydig cell tumors would still be increased after adjusting for differences in survival, but the formal statistical analysis was too complex to complete during the meeting.

The panel discussed the role of peroxisome proliferation as the mode of action of Leydig cell tumors. Specifically, the panel discussed a workshop publication **(**Clegg et al. 1997**)** that evaluated the seven known modes of action for Leydig cell tumors. Most of the modes of action involve altered hormonal response in response to peroxisome proliferation, including increased estradiol via hepatic aromatase and binding to the TGF α receptor or elevations in leutinizing hormone to compensate for the testes becoming less responsive to this hormone. One panelist emphasized that the monkey study (Thomford et al., 2001) showed no effects in the testes, even though the animals were dosed at C8 levels high enough to cause major weight loss and mortality. This panelist suggested that this indicates the Leydig cell effects seen in rats are unlikely to occur in primates. This panel member also noted that no increased estradiol was noted in the monkeys.

One panelist observed that Leydig cell tumors were a classic response to peroxisome proliferation but the available studies do not provide positive evidence, such as increased estradiol levels, that peroxisome proliferation is the operative mode of action. The panelists agreed that while data gaps exist, a peroxisome proliferation mode of action was a reasonable assumption. One panelist stated that whatever the MOA was, it was not genotoxicity.

The panel agreed unanimously that for Leydig cell tumors:

- All 7 possible mechanisms for Leydig cell tumors are non-linear; therefore a non-linear dose-response approach is reasonable;
- Humans have a low incidence of these tumors;
- The monkey study did not demonstrate Leydig cell pathology or increased estradiol;
- Leydig cell tumors are a known tumor type for other peroxisome proliferators;
- Humans do not develop Leydig cell tumors following exposure to other known peroxisome proliferators such as clofibrate;
- Regardless of the actual mode of action, it is likely to be non-genotoxic.

Outcome: The panel agreed that based on the absence of genotoxicity, the Leydig cell tumors were likely to be caused by a non-genotoxic mechanism. The panel further agreed that if sufficient evidence were available to show increased estradiol levels (i.e., secondary to peroxisome proliferation) as the mechanism for the observed tumors, then the mechanism would be non-genotoxic and would not be quantitatively similar or possibly not relevant at all to humans. However, without this evidence this effect can not be totally discounted.

Pancreatic tumors

Key Panel Discussion Points: Since the tumor results from the Beigel et al., (2001) were not provided in the summary table distributed to the panel prior to the meeting, the pancreatic tumor data from this study were presented as a table at the meeting (see Table 7 below):

Table 7 Biegel Study: Pancreas Tumors			
	Control	pair-fed control	300 ppm
Hyperplasia	14/80 (18%)	8/79 (10%)	30/48* (40%)
Adenomas	0/80	1/79	7/76*
Carcinomas	0/80	0/79	1/76

One panelist described an analysis that had been done to compare the two cancer studies with regard to the pancreatic tumors. This panelist noted that although the first study (Riker Laboratories, 1983) did not report pancreatic tumors or hyperplasia, the second study (Biegel et al., 2001) did. However, this panel member also noted that the studies were not inconsistent because of the different definitions of adenoma versus hyperplasia based on pancreatic cell size used by the respective investigators. Also, the criteria for separating hyperplasia from adenomas is based on lesion size. Both studies were qualitatively similar with a number of larger lesions (adenomas) found in the Biegel study. Another scientist commented, when the two studies were recently compared by a group of pathologists using current criteria, there was a consistency in a pancreatic response; however, there was not an increased number of adenomas found in the earlier study. Instead, an increase in hyperplastic nodules of the acinar pancreas was found, which is consistent with the Beigel study. However, even though the dietary dose was the same (300 ppm), the Riker Laboratories study rats did not develop these hyperplasias into adenomas to the extent that occurred in the Beigel study.

With regard to the potential mode of action, one panelist suggested that the persistent increase seen in cholecystokinin and increased bile acids may be involved in the MOA, but the evidence in rats, monkeys and humans does not support this hypothesis. When a panelist asked if a strong case could be made that the pancreatic tumors resulted from peroxisome proliferation, several panelists responded no. Another added that while some peroxisome proliferation agents cause the triad of tumors seen with C8, not all do. Another panelist added that no pancreatic, liver, or testes hyperplasia was noted in monkeys at the time of sacrifice.

Outcome: The panel agreed that the evidence was not sufficient to demonstrate the MOA for pancreatic tumors, but enhanced cell proliferation (hyperplasia) was likely to be involved. The MOA appears to be non-genotoxic based on the results of genotoxicity bioassays.

Mammary Fibroadenomas

Key Panel Discussion Points: The panel considered whether the fibroadenomas observed in the Riker Laboratories study were a real treatment-related effect, or an artifact of classification, since other mammary tumor types observed in this study showed no clear relationship with dose. Table 8 below shows the data for several types of mammary tumors from this study:

Table 8. Riker Study: Mammary Tumors			
	Control	30 PPM	300 PPM
Adenomas	7%	0	0
Adenocarcinomas	15%	31%	11%
Carcinomas	2%	0	0
Fibroadenomas	22%	42%	48%*

One panelist suggested that even though fibroadenomas were statistically significant, when all mammary tumor types are combined, they are not likely to be significant. It was noted by the panel that the individual incidence data from the study would need to be examined to determine the combined incidence of all mammary tumor types, rather than adding the percentages from each category. The panel discussed the histological basis for reporting separately fibroadenomas versus other types of mammary adenomas. A panelist suggested that since fibroadenomas do not progress to the other types it is correct to report them separately. Another said that the National Toxicology Program (NTP) reports fibroadenomas combined with adenomas.

The panel also discussed potential modes of action for mammary tumors. Increased estradiol was proposed as a possible MOA for the induction of hyperplasia and tumor formation, but the panel did not believe the data were sufficient to demonstrate this proposed mode of action. A panelist asked if a linear assessment could be done to help decide the importance of the effect. Another responded that the data were not adequately fit by any of the acceptable dose-response models, so a quantitative dose-response assessment was not reported for this data set.

Outcome: The panel agreed the data are not adequate to demonstrate a MOA; however based on the negative genotoxicity assays, C8 is unlikely to be genotoxic. Several panelists were not convinced the data demonstrated any real tumorigenic effect.

Cancer Dose-Response Assessment

After evaluating the relevance of each tumor type to humans, and the potential mode of action, the panel members were asked to recommend a dose-response approach for each tumor type. In all cases the panel agreed unanimously unless noted otherwise. For the liver tumors, the panel agreed that the MOE approach was most appropriate. For the remaining tumor types, the panel agreed that both linear and MOE approaches were appropriate, since the mode of action was not considered to have been adequately demonstrated for any of these three tumor types. All panel members agreed with these conclusions, except for the Leydig cell tumors, where one panel member argued that only an MOE approach should be used.

For the liver tumors, the MOE approach was selected. Since the MOE analysis often uses the benchmark response for a precursor as the basis of deriving a point of departure, the panel judged the pRfD for liver effects as sufficiently protective of potential liver carcinogenicity.

For Leydig cell tumors, benchmark dose modeling was conducted to identify a point of departure for the linear and MOE assessments. The Point of Departure (POD) for Leydig cell was chosen by the panel from the BMD modeling output. The BMDL of 0.32 mg/kg-day was selected as the most appropriate basis for deriving the assessment.

The panel discussed the appropriate factors to apply to the BMR for completing the MOE assessment. The panel noted that EPA's 1999 guidelines have only recently begun to be applied, and that formal guidance or examples of the interpretation and default values to use in deriving the MOE are lacking. In discussing the important considerations for the MOE, the panel decided that the critical factors to be considered were for "Nature of Effect", Intrahuman sensitivity" and "Animal to Human Extrapolation". A summary of the factors chosen is shown in Table 9.

For the Leydig cell tumors, a factor of 3 for nature of effect was selected as the most appropriate value, since the observed effect was for benign tumors. A factor of 10 was selected for Intrahuman sensitivity. A factor of 3 was used for Animal to Human Extrapolation, since dosimetric adjustments were applied to the dose data used for the BMD modeling. This composite factor of 100 was further supported since these types of tumors, although common in rats, are found rarely in people. In addition, the mode of action is likely via peroxisome proliferation which is quantitatively much less important in humans. The panel agreed that the composite MOE of 100 was appropriate.

For the linear dose-response assessment for Leydig cell tumors the BMDL of 0.32 mg/kg-day was used to calculate an oral cancer slope factor as follows:

Slope factor = risk/dose = 0.1/0.32 = 0.31 per mg/kg-day

(Note: risk is numerically expressed as 0.1 because the BMDL is the point that represents a 10% increased in tumor incidence in accordance with EPA guidance.) BMD modeling failed for the tumor data for pancreatic tumors and mammary gland fibroadenomas. Therefore, the panel determined that the data for these two tumor types were not adequate to conduct a quantitative dose-response assessment.

Table 9.
Factors Used to Describe Various Areas in the Development of MOEs for Cancer Endpoints.

Tumor	Model	Nature Of Effect	Intra Human	Animal to Human	Steepness of Slope	Total Exposure	MOE
Liver	MOE	1	10	10	NR	NR	100
Leydig	both	3	10	3	NR	NR	100
Pancreas	both	NA (cannot be modeled)					
Mammary	both	NA (cannot be modeled)					

NR = Not Relevant based on panel judgment; NA = Not Applicable

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The panel also voted on confidence ratings for the cancer assessment. *TERA* noted that according to EPA guidance "high confidence" suggests that the assessment is unlikely to change with the availability of new data, while "low confidence" indicates that the assessment is likely to change with new data. Based on these criteria the panel voted on their confidence in the cancer assessment using either the pRfD for liver toxicity to adequately account for the liver cancer risk or using the assessment based on Leydig cell tumors. The panel voted as follows:

Liver pRfD = high (7 votes); medium-high (2 votes)
Leydig tumors = low (7 votes); low-medium (2 votes)

Therefore, the panel agreed that the oral pRfD for liver toxicity would be the basis for determining water and soil screening levels (which are based primarily on oral exposure) for the following reasons:

- high confidence in the pRfD (i.e., not likely to change in the future due to additional data collection);
- the pRfD would be protective against the quantitatively less sensitive and questionable relevance peroxisome proliferation-related liver cancer in humans;
- low confidence in the Leydig tumor analysis and questionable relevance to humans;
- limitations in study design, data quality, and data interpretation rendered difficult the determination of whether the reported increased incidence of pancreatic tumors or mammary tumors were related to C8 treatment, and did not allow the modeling of a point of departure that could serve as the quantitative basis for risk value development.

Screening Levels

(Note: Dr. Seed was absent during this part of the meeting)

The consent order required that screening levels be developed for drinking water, soil, and air. The panel followed the guidance provided by U.S. EPA's "Risk Assessment Guidance for Superfund" as further explained by both Region 3 and Region 9 risk-based concentration guidance. In cases where a conflict occurred between the guidance documents, Region 9 guidance was followed because it is more conservative, i.e. more health protective. For drinking water and soil, only ingestion and dermal absorption were considered as routes of exposure. EPA guidance indicates volatilization from water or soil should only be evaluated for chemicals with Henry's law constants greater than 10^{-5} and molecular weights less than 200. Since C8's Henry's Law constant is 10^{-11} and its molecular weight is 431, volatilization was not evaluated.

As discussed above, the panel concluded that since both liver and Leydig cell tumors were potentially formed via nonlinear modes of action, and further since greater confidence was placed in the quantitative assessment based on the liver endpoint, the pRfD and pRfC for liver toxicity would be protective of potential cancer effects of C8. The panel considered that the linear extrapolation for Leydig cell tumors was too uncertain to be used with confidence and that the MOE approach based on the Leydig cell tumors gave essentially the same numerical value as that for the liver endpoint, but with less confidence. Thus, the pRfD and pRfC for liver toxicity, and "noncancer" equations were used for calculating screening levels. Screening levels are calculated following the premise that if lifetime exposure is equal to or less than the pRfD or pRfC, then no risk of deleterious effects is expected. Mathematically, this concept can be expressed by the following standard equation; the ratio of the measured or estimated exposure to the RfD is called the Hazard Quotient.

If Exposure ÷ RfD = 1 or less, then no risk of deleterious effects is presumed.

Using this concept, it is possible to estimate the concentration in media that results in a lifetime exposure equal to the pRfD or pRfC. These equations, from EPA Region 9's guidance on deriving risk based concentrations, are listed below:

Air Screening Level : $[\ ]\ ug/m^3 = \frac{THQ \times RfDi \times BW \times AT \times 1000}{EF \times ED \times \text{air IR}}$

Note: $RfDi\ (mg/kg\text{-}day) = RfC \times \frac{20m^3/d\ (IR)}{70\ kg\ (BW)}$

Soil Screening Level: $[\ ]\ mg/kg = \frac{THQ \times AT \times BW}{EF \times ED \times [\text{soil IR} / RfD \times 10^{-6} + SA \times AF \times ABS / RfD \times 10^{-6}]}$

Water Screening Level: $[\ ]\ ug/L = \frac{THQ \times AT \times BW \times 1000}{EF \times ED \times [\text{water IR} / RfD]}$

Where:

THQ	=	Target Hazard Quotient, assumed to be 1
RfDi	=	The RfC expressed in terms of dose, mg/kg-day
RfD	=	The oral reference dose estimated by the panel, 0.004 mg/kg-day
RfC	=	The inhalation reference concentration estimated by the panel, see below
BW	=	Body weight, assumed to be 70 kg for adults and 15 kg for children
AT	=	Averaging time, 10950 days, the exposure duration expressed in days
EF	=	Exposure Frequency, 350 days/year, the average number of days each year people are exposed
ED	=	Exposure duration, 30 years, the average number of years people are exposed
IR	=	Inhalation rate for air screening levels, 20 m^3/day; Ingestion rate for soil and, Water screening levels, 200 mg/day soil ingested based on child exposure and, 2 L/day water ingested based on adult exposure
SA	=	Surface area of exposed skin, 2800 cm^2/day
AF	=	Adherence factor, 0.2 mg/cm^2, the amount of soil that adheres to skin
ABS	=	Skin absorption factor, specific factor not available for C8, assumed to be 0.1 for semi-volatile chemical per EPA guidance

The panel unanimously agreed that the equations, assumptions, and default exposure parameters described above were the appropriate choices for calculating screening levels for air, soil, and water. The following values are the screening levels estimated by the equations.

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For air: 0.1–6.0 micrograms per cubic meter of air ($\mu g/m^3$) ambient air. Note that the panel considered this range to be interim until the additional work discussed for the RfC is completed. This range incorporates the range of possible $NOAEL_{HEC}$s estimated by *TERA* prior to the meeting as well as the range of composite uncertainty factors recommended by the panel. The final pRfC is discussed in the following section Post Meeting Action Items.

For soil: 244 miligrams per kilogram of soil (mg/kg) residential soil, rounded to 240 mg/kg.

For water: 146 micrograms per liter of water (µg/L), rounded to 150 µg/L.

2.3 POST MEETING ACTION ITEMS

The following activities were conducted after the CATT Toxicologists meeting.

Derivation of the pRfC for C8

The CATT panel could not develop a final recommendation on the pRfC or the air screening level during the May 6 and May 7, 2002 meeting. This was due to a lack of data necessary for these calculations. At the meeting, the panel chose the key study for risk factor derivation as the 2-week inhalation study by Kennedy et al. (1986) and voted upon the uncertainty factors. They directed the author, panel member Kennedy (DuPont), to (1) retrieve the standard deviation data for the absolute and relative liver weight data sets; and (2) to measure the particle size distribution in the exposure chamber and determine the corresponding standard deviation; and (3) to provide these data to DEP and to *TERA*. The panel directed *TERA* to utilize these data to develop the pRfC based on the most sensitive organ (liver or lung) and the air screening level based on USEPA Region 9 standard formulas.

During the meeting, the CATT panel agreed that the Kennedy et al. (1986) study was the most appropriate basis for deriving the pRfC, with the developmental study by Staples et al. (1981) providing support for the selected critical effect levels. The CATT panel identified a NOAEL for increased liver weight at the lowest study concentration of 1.0 mg/m^3, with a LOAEL of 7.6 mg/m^3. The NOAEL for lung effects was identified by the CATT panel as 7.6 mg/m^3, with a LOAEL was 84 mg/m^3.

In order to derive an pRfC, the reported study concentrations were converted to human equivalent concentrations ($Conc_{HEC}$), according to current U.S. EPA RfC methodology (USEPA, 1994). The calculation of the $Conc_{HEC}$ requires two steps. First, the study concentration is adjusted from the exposure duration used in the experiment to an equivalent continuous exposure concentration ($Conc_{ADJ}$). Animals in this study were dosed for 6 hours per day, for five days, then not dosed for two days, and dosed again for five days and sacrificed at the end of the 12^{th} day; hence, continuous exposure duration adjustment was made as follows:

$$\text{Study concentration} \times (6 \text{ hours}/24 \text{ hours}) \times (10 \text{ days}/12 \text{ days}) = Conc_{ADJ}$$

Second, the duration-adjusted concentrations ($Conc_{ADJ}$) were converted to human equivalent concentrations ($Conc_{HEC}$) to account for differences in the respiratory tract anatomy and physiology for the test species versus humans. This conversion is made as follows:

$$Conc_{ADJ} \times RDDR = Conc_{HEC}$$

The RDDR is the Regional Dose Deposition Ratio calculated using U.S. EPA's RDDR software program (USEPA, 1994). The RDDR depends on the characteristics of the particle size distribution (e.g., mass median aerodynamic diameter, and geometric standard deviation), the test species and body weight, and the region of the respiratory tract (or extrarespiratory tissue target if applicable) affected by exposure. Appropriate particle size characteristics to use as inputs into the RDDR software were obtained from a recent communication from DuPont (see attached). For the Kennedy et al. (1986) study, the test sex and species was male rats. Since body weight data were provided in the study, these data were used directly in the RDDR program. The mean body weight data on day 5 of exposure was used for this calculation, rather than the study-day 10 body weight data. The day 5 body weights were

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used because there was evidence of changes in body weight over the 12-day study period, and therefore, this value was judged as the best estimate of the mean body weight over the period of exposure.

The CATT panel considered two potential critical effects for deriving the pRfC; increased liver weight and overt toxicity secondary to pulmonary toxicity. The RDDR for extrarespiratory tissues was the most appropriate value to use in calculating human equivalent concentrations for assessing the liver effects. The RDDR program calculates values for a variety of different regions of the respiratory tract. The CATT panel agreed that the overt toxicity of C8 was likely due to particle overload, as supported by pulmonary edema in the acute study reported in the same paper (Kennedy et al., 1986). Therefore, the RDDR for the pulmonary region was selected as most appropriate respiratory tract region for calculating the human equivalent concentrations. The calculation of the human equivalent concentrations used in the dose-response assessment is summarized in Table 10.

Table 10. Calculation of Human Equivalent Concentrations for Kennedy et al. (1986)

		Extrarespiratory		Pulmonary	
Study Concentration[a]	$Conc_{ADJ}$	RDDR[b]	$Conc_{HEC}$	RDDR	$Conc_{HEC}$
1.0	0.21	2.956	0.62	0.513	0.11
7.6	1.6	2.954	4.7	0.512	0.81
84	17	2.973	52	0.521	9.1
a. All concentrations reported in the table are in units of mg/m^3.					
b. The RDDR values are taken from the EPA RDDR Program Output provided in the attachment					

Benchmark Concentration Modeling

The CATT panel further recommended that benchmark concentration (BMC) modeling be performed for the increased liver weight endpoint from the Kennedy et al. (1986) study. The published version of the study did not provide standard deviations to accompany the group mean data, and therefore, BMC modeling could not be performed at the time of the CATT panel meeting. Subsequent to the meeting, the individual liver weight data for this study were obtained from DuPont (see attached). The individual animal data were used to calculate group mean and standard deviations. These data were then employed for the BMC analyses.

The modeling was conducted according to draft EPA guidelines (U.S. EPA, 2000) using Benchmark Dose Software (BMDS version 1.3.1), available from the U.S. EPA website (U.S. EPA, 2002). The endpoints of interest with respect to C8 liver toxicity were continuous rather than quantal (e.g., incidence data) in nature. Therefore the absolute and relative liver weight data sets were modeled using the linear, Hill, power, and polynomial models. An acceptable fit to the data was defined as a goodness-of-fit p-value greater than or equal to 0.1, or a perfect fit when there were no degrees of freedom for a formal statistical test of fit. Choice of 0.1 is consistent with current U.S. EPA guidance for BMD modeling (U.S. EPA, 2000). Goodness-of-fit statistics are not designed to compare different models, particularly if the different models have different numbers of parameters. Within a family of models, adding parameters generally improves the fit. BMDS reports the Akaike Information Criterion (AIC) to aid in comparing the fit of different models. When comparing the fit of two or more

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models to a single data set, the model with the lesser AIC was considered to provide a superior fit. The benchmark response (BMR) level used for this analysis was set at a standard deviation (SD) value of 1.0. This value was chosen based on EPA draft guidelines for BMC analysis (U.S. EPA, 2000), in the absence of a clear biological rationale for selecting an alternative response level.

The following guidance was followed with regard to the choice of the Benchmark Concentration Lower Limit (BMCL) to use as a point of departure for calculation of the pRfC. This guidance is consistent with recommendations in U.S. EPA's BMC guidance (2000). For each endpoint, the following procedure is recommended:

1. Models with an unacceptable fit are excluded.

2. If the BMCL values for the remaining models for a given endpoint are within a factor of 3, no model dependence is assumed, and the models are considered indistinguishable in the context of the precision of the methods. The models are then ranked according to the AIC, and the model with the lowest AIC is chosen as the basis for the BMCL.

3. If the BMCL values are not within a factor of 3, some model dependence is assumed, and the lowest BMCL is selected as a reasonable conservative estimate, unless it is an outlier compared to the results from all of the other models. Note that when outliers are removed, the remaining BMCLs may then be within a factor of 3, and so the criteria given in item 2 would be applied.

4. The BMCL values from all modeled endpoints are compared, along with any NOAELs or LOAELs from data sets that were not amenable to modeling, and the lowest NOAEL or BMCL is chosen.

The BMC results are summarized in Table 11 and the individual BMDS model run output is provided in the attachments.

For modeling of the absolute liver weight data set, a constant variance model was appropriate (see test 2 in the BMDS output). The power and polynomial models both defaulted to a linear model. None of these linear models fit the data well. The Hill model provided an excellent fit to the data, as indicated by visual inspection of the fit and the comparison of the maximum likelihood estimates for the fitted model to the optimum model (shown as model A1 in the BMDS output). The linear models failed to provide an adequate fit to the full data set, since they did not accommodate the plateau of the concentration-response curve between the mid- and high-concentrations. BMC modeling was redone using a truncated data set (high concentration group removed) to optimize the fits of these models. Removing the high concentration resulted in good fits for the linear models (the power and polynomial models again defaulted to linear) as indicated by the AIC and goodness-of-fit p-values. The Hill model could not be run with the truncated data set since at least four concentration groups are required to provide a model fit.

Adequate fits to the data were achieved when the high concentration data were removed. An argument could be made for using these results as the best estimate for the data set, since an adequate fit was achieved with fewer parameters than for the Hill model using the full data set. However, the BMCL estimate for the full data set was on the border of 3-fold lower than for the truncated data set, which would suggest that the lower BMCL should be selected. Furthermore, comparison of the chi square residuals in the range of the NOAEL concentration suggests that the Hill model provided a better fit of the data in the low concentration region than the linear models using the truncated data. Finally, since

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there was no biological rationale for removing the high concentration data from the modeling, an adequate model fit for the full data set is preferred over the model fit for the truncated data set. Based on these considerations, the BMC of 0.78 mg/m^3 and corresponding BMCL of 0.33 mg/m^3 are considered the best estimates for the absolute liver weight data set.

The relative liver weight data displayed a similar plateau between the mid- and high-concentration groups. The linear, power, and polynomial models all failed to provide an adequate fit. As for the absolute liver weight data, the Hill model provided an excellent fit to the data, but in this case failed to calculate a BMCL. In the absence of an adequate BMCL estimate for any of the models using the full data set, the data were remodeled with the high concentration group data removed. The power and polynomial models were nearly linear, as indicated by the parameter estimates in the BMDS output. The linear, power, and polynomial models all provided a similar, and very good visual fit to the data. The goodness-of-fit statistic for the linear model was 0.9. Although BMDS did not calculate the goodness-of-fit p-values for the power and polynomial models, inspection of the maximum likelihood estimates for these fitted models as compared to the optimum model (model A1 in the BMDS output) confirmed the good fit. The linear model provided a similar BMC and BMCL estimate as the power and polynomial models, but required less parameters to do so (i.e., as reflected in the lower AIC). Therefore, the BMC of 1.3 mg/m^3 and the corresponding BMCL of 0.94 mg/m^3 are considered the best estimates for the data set for relative liver weight.

At the time of the meeting the CATT panel did not provide a recommendation on whether absolute or relative liver weight should be considered more appropriate as the critical effect. Both of these measures were significantly increased beginning in the 7.6 mg/m^3 study concentration group. One would not expect a difference in the sensitivity of these two measures in this case, because there was no change in body weight (the basis for calculating relative liver weight) at the NOAEL. Therefore, both absolute and relative liver weight changes are considered to be an adequate basis for the critical effect. Based on this consideration, the lower of the BMCL estimates for the absolute and relative liver weight changes is the most appropriate basis for deriving the pRfC. The BMC of 0.78 mg/m^3 with the corresponding BMCL of 0.33 mg/m^3 for increased absolute liver weight are the best estimates from the BMC modeling results. The BMCL of 0.33 mg/m^3 is the most appropriate choice as the critical effect level for derivation of the pRfC, because the BMCL is lower than either the NOAEL of 0.61 mg/m^3 for liver effects or the NOAEL of 0.81 mg/m^3 for pulmonary effects in this study.

Selection of uncertainty factors

As described in the technical meeting notes, the CATT panel unanimously agreed on the choice of 3 for extrapolation from an animal study (UF_A), a factor of 10 to account for variability in human sensitivity (UF_H), and a factor of 1 for extrapolation from study NOAEL or BMDL (UF_L). The CATT panel considered the selection of U.S. EPA's other two factors, for extrapolation from a study of less-than-lifetime duration (UF_S) and for database insufficiencies (UF_D), to be dependent on whether liver or lung was ultimately selected as the critical effect. The panel was not unanimous in selection of the UFs or UF_D for either organ, but a clear majority vote was obtained for these UFs regarding liver toxicity.

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Based on the liver as a critical effect, panel members recommended values of either 1 (one vote), 3 (six votes) or 10 (1 vote) for UF_S, and values of 3 (six votes) or 10 (two votes) for UF_D. Therefore, based on the liver as the critical effect, the composite UF would range from 100 to 1000, depending on the selection of the values for UF_S and UF_D. The majority vote of the CATT panel (Table 5) supported a factor of 3 for UF_S and 3 for UF_D. Based on these values, a composite UF of 300 for liver effects was calculated.

Based on the lung as the critical effect, panel members recommended values of either 1(three votes), 3 (three votes) or 10 (two votes) for UF_S, and values of 1 (one vote), 3 (five votes), and 10 (two votes) for UF_D. Therefore, with the lung as the critical effect the composite UF would range from 30 to 3000. The majority of the CATT panel supported a value of 3 for UF_D based on lung effects. A clear majority vote was not determined for any one value for the UF_S; however, six votes were cast for a value lower than 10 and five votes were cast for a value higher than one; thus the median value of 3 would be a reasonable choice. Therefore, values of 3 for both UF_D and UF_S for lung effects would also result in a composite UF of 300.

However, it is important to note that the panel could not arrive at a consensus on the overall magnitudes of UF_S and UF_D, because of the numerous uncertainties with the inhalation database. The resulting range in the uncertainty factor was generally considered reasonable by the panel, with values falling within this range being indistinguishable from each other.

Calculation of the pRfC

Liver toxicity was identified as the critical effect because it was more sensitive to C8 than the lung (i.e., liver toxicity had a lower NOAEL or BMCL than lung), the composite UF ranged from 100 to 1000 and was 300 based on the majority vote.

The pRfC is calculated as follows:

$$\text{pRfC } (mg/m^3) = \text{critical effect level} \ / \ \text{composite UF}$$

$$\text{pRfC range} = 0.33 / 1000 = 0.00033 \text{ mg/m}^3 \text{ (or rounded to 0.3 } \mu g/m^3)$$

to

$$= 0.33 / 100 = 0.0033 \text{ mg/m}^3 \text{ (or rounded 3.3 } \mu g/m^3)$$

$$\text{pRfC (majority vote)} = 0.33 / 300 = 0.0011 \text{ mg/m}^3 \text{ (or rounded to 1 } \mu g/m^3)$$

Therefore, the recommended pRfC based on the majority vote for a composite UF of 300 is 1 microgram per cubic meter of air ($\mu g/m^3$) with a range from 0.3 $\mu g/m^3$ to 3.3 $\mu g/m^3$.

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Table 11. Benchmark Dose Modeling Results for C8[a]				
Model/Data Set	**AIC**	**P-value**	**BMC[b]**	**BMCL**
Absolute Liver Weight –All Data Modeled				
Linear	62.58[c]	<0.001[d]	31	19
Hill	48.67	1.0[e]	**0.78**	**0.33**
Power	62.58[c]	<0.001	31	19
Polynomial	62.58[c]	<0.001	31	19
Absolute Liver Weight - High Concentration not Modeled				
Linear	38.22[c]	0.72	1.6	1.1
Power	38.22[c]	0.29[d]	1.6	1.1
Polynomial	38.22[c]	0.72	1.6	1.1
Hill	Insufficient Number of data points to run model			
Relative Liver Weight – All Data Modeled				
Linear	-167.65[c]	<0.001	21	15
Hill	-184.29	1.0[e]	1.1	Failed
Power	-167.65[c]	<0.001	21	15
Polynomial	-167.65[c]	<0.001	21	15
Relative Liver Weight - High Concentration not Modeled				
Linear	-137.04[c]	0.90	**1.3**	**0.94**
Power	-135.05[c]	Failed	1.5	0.94
Polynomial	-135.05[c]	1.0[e]	1.5	0.94
Hill	Insufficient Number of data points to run model			

[a] Modeling was performed based on absolute and relative liver weight results reported in Kennedy et al. (1986).

[b] BMC and BMCL are based on benchmark response of 1SD. Results are presented in units of mg/m^3. BMC and BMCL estimates in bold type are the estimates judged to be the best estimates for each endpoint. "Failed" indicates that BMDS was unable to produce the estimate or the information required to be able to present a value.

[c] Corrected from erroneous BMDS output. Errors were identified in the degrees of freedom (DF) provided in the output for the fitted model in several cases. For these cases, the AIC was calculated independently using the log likelihoods provided in the output and the correct number of DF. Similarly, the goodness-of-fit p-values were corrected by calculating manually the chi square p-value using the appropriate number of DF.

[d] This model provided an identical fit to the linear and polynomial models. The reported P-value reflects a difference in the maximum likelihood estimate for the comparison model (Model A1 in the BMDS output) across the three models. This difference the maximum likelihood estimate should be the same for all three models, since this estimate is model independent.

[e] A fit that maximizes the likelihood is assigned a p-value of 1.0, even if there were no degrees of freedom for a formal statistical test. The maximized likelihood is given by model A1 for constant variance models and model A2 for non-constant variance models. Models A1 and A2 are independent of the model chosen to fit the data (e.g., power, polynomial, Hill model) and provide the best match possible to the mean and standard deviation for each dose level.

Calculation of an Air Screening Level

As described in the technical meeting notes, U.S. EPA Region 9 methodology was judged by the CATT panel to be an appropriate basis for deriving the air screening level. The following standard formula was used to calculate the air screening level:

$$\text{Air Screening Level } (\mu g/m^3) = \frac{\text{THQ x RfDi x BW x AT x 1000}}{\text{EF x ED x air IR}}$$

$$\text{Note: RfDi (mg/kg-day)} = \text{RfC x } \frac{20 m^3/d \text{ (IR)}}{70 \text{ kg (BW)}}$$

Where:

THQ	=	Target Hazard Quotient, assumed to be 1
RfDi	=	The RfC expressed in terms of dose, mg/kg-day
RfC	=	The inhalation reference concentration (mg/m^3)
BW	=	Body weight, assumed to be 70 kg for adults
AT	=	Averaging time, 10,950 days, the exposure duration expressed in days
EF	=	Exposure Frequency, 350 days/year, the average number of days each year people are exposed
ED	=	Exposure duration, 30 years, the average number of years people are exposed
IR	=	Inhalation rate for air screening levels, 20 m^3/day

Using this equation, the air screening level ranges from 0.3 $\mu g/m^3$ to 10 $\mu g/m^3$. Using a reasonable median value, the air screening level would be 1.1 $\mu g/m^3$ (or rounded to 1 $\mu g/m^3$).

2.4 SUMMARY OF FINDINGS

The key studies, critical effects and levels, uncertainty factors, and provisional risk factors developed by the CATT toxicologists are summarized in Table 12.

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Table 12. Summary of RfD and RfC Values for C8 Determined by the CATT Toxicologists									
Reference	Critical Effect	Critical Effect Level[a]	UF_A	UF_H	UF_L	UF_S	UF_D	Composite UF[b]	RfD/RfC
Oral Studies									
Palazzolo et al. (1993)[c] 90-day rat study	Increased relative liver weight with histopathology in male rats	0.47 (NOAEL in males) 0.72 (BMDL)	10	10	1	1	1	100	0.005 0.007
York et al. (2002) Two-Generation rat study	Increased liver weight in male rats, supported by histopathology at higher doses (histopathology was not examined at the lowest dose, but incidence of hypertrophy was 100% at next highest dose).	0.42 (BMDL in males)[d]	10	10	1	1	1	100	0.004
RikerLaboratories (1983) Two-year rat study	Hepatic megalocytosis in male rats.	0.73 (BMDL in males)	10	10	1	1	1	100	0.007
Thomford et al. (2001)[e]26-week cynomolgus monkey study	Decreased thyroid hormone levels in male cynomolgus monkeys, and supported by a NOAEL at the same dose for clinical signs of toxicity in the co-critical rhesus monkey study (Goldenthal et al., 1978b)	3 - 10 (LOAEL in males)	10	10	3	3	1	1000	0.003 - 0.01

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Inhalation Studies									
Kennedy et al. (1986)[f] Two-week rat study	Increased liver weight supported by histopathology and clinical chemistry in male rats	0.61(NOAEL - HEC_{ER}males) 0.33 (BMCL, BMC 0.78 absolute liver weight) 0.94(BMCL, BMC 1.3 relative liver weight)	3	10	1	3	3	300	1
Dermal Studies									
Kennedy et al. (1985)[f] Two-week rat study	Increased liver weight in male rats	4.2[g] (LOAEL in males)							Data Inadequate

a. Oral and Dermal effect levels and RfDs are presented in units of mg/kg-day, while the inhalation critical effect level and RfC is presented in units of mg/m^3

b. Areas of uncertainty addressed by uncertainty factors are: animal to human extrapolation (A); intrahuman variability and protection of sensitive subpopulations (H); extrapolation from a LOAEL to a NOAEL(L); extrapolation from a subchronic to chronic exposure (S); and lack of a complete database (D)

c. The subchronic study by Goldenthal et al. (1978a) could serve as a supporting study for liver effects in rats.

d. BMDL is the 95% lower confidence limit on the dose corresponding to a response level of 10% or an increase of 1SD in the continuous endpoint being assessed. Only modeling results that provided the lowest value and provided an adequate fit to the data are provided.

e. The subchronic study in rhesus monkeys by Goldenthal et al. (1978b) is a co-critical study for clinical signs of toxicity in monkeys.

f. These studies are not adequate for derivation of an IRIS quality RfD/RfC of even low confidence. The values shown could be used to derive a provisional value. Derivation of the RfC or RfD via route-to-route extrapolation is not supported by the available toxicokinetic data. Consensus on the values for UF_S and UF_D was not reached by the panel; however, a majority vote was obtained for a value of 3 for both these UFs in reference to liver as the target organ. See text of this report for ranges of UFs and SLs based on the range distribution of the votes for UFs.

g. 4.2 mg/kg-day reflects the study dose of 20 mg/kg adjusted for discontinuous exposure.

I agree that the notes as presented accurately reflect the panel's discussion and conclusions during the May 6-7, 2002 C8 Assessment of Toxicity Toxicologists Panel Meeting, and that the post meeting actions taken to develop the pRfC and Air Screening Level are in accordance with the instructions provided to *TERA* by the panel. (Original signatures are on file at DEP.)

John Cicmanec, D.V.M., M.S., ACLAM, USEPA ORD	Date
Joan Dollarhide, M.S., M.T.S.C., J.D., *TERA*	Date
Michael Dourson, Ph.D., D.A.B.T., *TERA*	Date
Gerald Kennedy, DuPont	Date
Andrew Maier, Ph.D., C.I.H., *TERA*	Date
Samuel Rotenberg, Ph.D., USEPA Region 3	Date
Jennifer Seed, Ph.D., USEPA Headquarters OPPT	Date
Dee Ann Staats, Ph.D., DEP (Chairperson)	Date
John Wheeler, Ph.D., D.A.B.T., ATSDR	Date
John Whysner, M.D., Ph.D., D.A.B.T.	Date

3. 0 COMPARISON OF SCREENING LEVELS TO SITE-RELATED DATA

After the SLs for air, water, and soil were determined, DEP compared these SLs to the site-related data that has been collected to date. These comparisons are summarized below. The work of the CATT was only one facet of an investigation that continues beyond the issuance of this report. The GIST is expected to issue a report of the groundwater and surface water data in early 2003. The air modeling effort continues and is currently focusing on determining the results of the air emissions reduction efforts by DuPont required in the consent order as a 50% reduction in overall emissions (both air and water) by the end of 2003. Upgrades were completed in June 2002 which included the installation of a new scrubber and increased height of the primary C8 emissions stack.

Water

To date, of the 188 samples collected from private wells, cisterns, and springs, 50 were used for drinking water and none exceeded the150 ppb health protective water SL for C8. Also to date, nine public water supply facilities in West Virginia have been analyzed for C8, including Belleville Locks and Dam, Blennerhassett Island, General Electric, Lubeck Public Service District (PSD), Mason County PSD, Parkersburg PSD, Racine Locks and Dam, New Haven Water Department, and Ravenswood. None of the drinking water from these facilities contained concentrations of C8 that exceeded the 150 ppb water SL. In fact, the concentrations of C8 in public water supplies were all below 2 ppb, below 15 ppb in private non-drinking water, and below 3 ppb in private drinking water wells in West Virginia. Samples were collected from Ohio public and private water supplies. Although C8 levels in some Ohio private water supplies were higher than those detected in West Virginia, none of these samples contained C8 concentrations above the water SL. These data have been provided to Ohio EPA and DEP will continue to share information with throughout the remainder of this investigation. The DEP notes that the water SL is higher than DuPont's internal community exposure guidelines for drinking water of 1 or 3 ppb; however, these guidelines were developed in the early 1990s and based solely on a two-week inhalation study from 1986. Since then significant additional toxicological data have been collected and the CATT water SL is based on a comprehensive examination of all available information. Sampling of the Ohio River has begun; preliminary analytical results are expected from the laboratory in September 2002. To date, no analysis has been performed to measure C8 in soils in West Virginia on private property; therefore, no comparison can be made to the soil SL.

Air

Mathematical computer models that incorporate weather conditions, chemical characteristics, and facility measurements were utilized by DEP to simulate the ambient air concentrations of C8. Based on actual emissions data from the DuPont WW facility for the year 2000, the DEP modeling efforts predicted a maximum C8 concentration in air of approximately 2.7 $\mu g/m^3$ at the facility fence line along the Ohio River. The maximum modeled C8 air concentration in the West Virginia residential area adjacent to the facility was approximately 0.2 $\mu g/m^3$ annual average. Predicted C8 air concentrations across the Ohio River from the WW facility in Ohio residential areas were greater than those predicted in residential areas in West Virginia. These data have been provided to Ohio EPA and DEP will continue to share information with Ohio EPA throughout the remainder of this investigation. Results of similar subsequent air modeling efforts conducted by DuPont are consistent with those of the DEP. Air modeling information can be obtained from the DEP Division of Air Quality.

The DEP's Divisions of Water Resources and Air Quality are currently reviewing all relevant air and water data to determine DuPont's compliance with the November 2001 consent order between DEP and DuPont.

ED_001803B_00003809-46

To: Jackson, Ryan[jackson.ryan@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Bowman, Liz[Bowman.Liz@epa.gov]; Baptist, Erik[baptist.erik@epa.gov]
From: Dourson, Michael
Sent: Mon 12/4/2017 1:05:10 PM
Subject: RE: Chlorpyrifos

Ryan

Ex. 5 - Deliberative Process

Mike

From: Jackson, Ryan
Sent: Monday, December 4, 2017 7:52 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>; Bowman, Liz <Bowman.Liz@epa.gov>; Baptist, Erik <baptist.erik@epa.gov>
Subject: Re: Chlorpyrifos

Ex. 5 - Deliberative Process

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Dec 4, 2017, at 7:50 AM, Dourson, Michael <dourson.michael@epa.gov> wrote:

> Ryan

Ex. 5 - Deliberative Process

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Jackson, Ryan
Sent: Monday, December 4, 2017 5:03 AM
To: Beck, Nancy <Beck.Nancy@epa.gov>

Cc: Bowman, Liz <Bowman.Liz@epa.gov>; Baptist, Erik <baptist.erik@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: Chlorpyrifos

Ex. 5 - Deliberative Process

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Nov 30, 2017, at 6:50 PM, Beck, Nancy <Beck.Nancy@epa.gov> wrote:

Attached for your review is an updated timeline for the chlorpyrifos evaluation.

Ex. 5 - Deliberative Process

A draft letter is attached.

Please let me know your thoughts on both.

Happy to chat.

Nancy

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

<Chlorpyrifos Timeline Draft.docx>

<LetterToColumbia_112817 RPK.docx>

To: Jackson, Ryan[jackson.ryan@epa.gov]; Lyons, Troy[lyons.troy@epa.gov]; Bowman, Liz[Bowman.Liz@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Tue 12/5/2017 11:27:13 PM
Subject: RE: Public Meeting Dec 6

Liz, Ryan and Troy

Ex. 5 - Deliberative Process

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

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From: Jackson, Ryan
Sent: Tuesday, December 5, 2017 1:00 PM

To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Bowman, Liz <Bowman.Liz@epa.gov>; Lyons, Troy <lyons.troy@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>
Subject: Re: Public Meeting Dec 6

Ex. 5 - Deliberative Process

Ryan Jackson

Chief of Staff

U.S. EPA

Ex. 6 - Personal Privacy

On Dec 4, 2017, at 12:47 PM, Dourson, Michael <dourson.michael@epa.gov> wrote:

Liz and Troy

Ex. 5 - Deliberative Process

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

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From: Bowman, Liz
Sent: Saturday, December 2, 2017 6:22 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Cc: Lyons, Troy <lyons.troy@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Jackson, Ryan <jackson.ryan@epa.gov>
Subject: Re: Public Meeting Dec 6

Ex. 5 - Deliberative Process

Sent from my iPhone

On Dec 1, 2017, at 9:19 PM, Beck, Nancy <Beck.Nancy@epa.gov> wrote:

> Thanks Troy. Looping in Ryan.
>
> **From:** Lyons, Troy
> **Sent:** Friday, December 1, 2017 9:09 PM
> **To:** Beck, Nancy <Beck.Nancy@epa.gov>
> **Cc:** Dourson, Michael <dourson.michael@epa.gov>; Bowman, Liz <Bowman.Liz@epa.gov>
> **Subject:** Re: Public Meeting Dec 6
>
> **Ex. 5 - Deliberative Process**
>
> Sent from my iPhone
>
> On Dec 1, 2017, at 9:04 PM, Beck, Nancy <Beck.Nancy@epa.gov> wrote:
>
> > Troy,

We are having a big public meeting on Wednesday Dec 6 on our new chemicals program. About 500 people have registered, 150 of those will be in person (the rest via webinar). Full range of stakeholders, including Hill staff who have been invited.

Ex. 5 - Deliberative Process

Thanks,
Nancy

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

To: Beck, Nancy[Beck.Nancy@epa.gov]
From: Dourson, Michael
Sent: Tue 12/5/2017 9:47:18 PM
Subject: Fwd: Follow-up from EFED 101

Sent from my iPhone

Begin forwarded message:

From: "Anderson, Brian" <Anderson.Brian@epa.gov>
Date: December 5, 2017 at 4:43:08 PM EST
To: "Dourson, Michael" <dourson.michael@epa.gov>, "Nesci, Kimberly" <Nesci.Kimberly@epa.gov>, "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>, "Keller, Kaitlin" <keller.kaitlin@epa.gov>
Cc: "Keigwin, Richard" <Keigwin.Richard@epa.gov>, "Echeverria, Marietta" <Echeverria.Marietta@epa.gov>
Subject: Follow-up from EFED 101

Hi Michael,

Ex. 5 - Deliberative Process

Please let me know if you need any more information or have any additional questions.

Thanks,

Brian

To: Camacho, Iris[Camacho.Iris@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Morris, Jeff[Morris.Jeff@epa.gov]; Mottley, Tanya[Mottley.Tanya@epa.gov]; Henry, Tala[Henry.Tala@epa.gov]; Barone, Stan[Barone.Stan@epa.gov]; Nguyen, Nhan[Nguyen.Nhan@epa.gov]; Selby-Mohamadu, Yvette[Selby-Mohamadu.Yvette@epa.gov]; Brinkerhoff, Chris[Brinkerhoff.Chris@epa.gov]; Scheifele, Hans[Scheifele.Hans@epa.gov]; Hanley, Mary[Hanley.Mary@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Schlosser, Paul[Schlosser.Paul@epa.gov]
Cc: Pierce, Alison[Pierce.Alison@epa.gov]; Hasan, Jafrul[Hasan.Jafrul@epa.gov]; Oxendine, Sharon[Oxendine.Sharon@epa.gov]; Fehrenbacher, Cathy[Fehrenbacher.Cathy@epa.gov]; Wolf, Joel[Wolf.Joel@epa.gov]; Brown, Judith[Brown.Judith@epa.gov]; Ortiz, Julia[Ortiz.Julia@epa.gov]
From: Dourson, Michael
Sent: Sat 12/2/2017 1:18:54 AM
Subject: RE: NMP Risk Evaluation (files part 2-Dupont 1990-file attached)

Iris

Thanks for all your extra effort here.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

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From: Camacho, Iris
Sent: Friday, December 1, 2017 6:17 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Mottley, Tanya <Mottley.Tanya@epa.gov>; Henry, Tala <Henry.Tala@epa.gov>; Barone, Stan <Barone.Stan@epa.gov>; Nguyen, Nhan <Nguyen.Nhan@epa.gov>; Selby-Mohamadu, Yvette <Selby-Mohamadu.Yvette@epa.gov>; Brinkerhoff, Chris <Brinkerhoff.Chris@epa.gov>; Scheifele, Hans <Scheifele.Hans@epa.gov>; Hanley, Mary <Hanley.Mary@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Schlosser, Paul <Schlosser.Paul@epa.gov>
Cc: Pierce, Alison <Pierce.Alison@epa.gov>; Hasan, Jafrul <Hasan.Jafrul@epa.gov>; Oxendine, Sharon <Oxendine.Sharon@epa.gov>; Fehrenbacher, Cathy <Fehrenbacher.Cathy@epa.gov>; Wolf, Joel <Wolf.Joel@epa.gov>; Brown, Judith <Brown.Judith@epa.gov>; Ortiz, Julia <Ortiz.Julia@epa.gov>
Subject: NMP Risk Evaluation (files part 2-Dupont 1990-file attached)

Attached is the DuPont 1990 study. This is the last file of the submission. Have a good weekend.

-Iris Camacho

****

Iris A. Camacho, Ph.D.

Senior Science Advisor (on detail)

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From: Camacho, Iris
Sent: Friday, December 01, 2017 5:37 PM
To: Beck, Nancy <beck.nancy@epa.gov>; 'Morris, Jeff' <Morris.Jeff@epa.gov>; 'Mottley, Tanya' <Mottley.Tanya@epa.gov>; 'Henry, Tala' <Henry.Tala@epa.gov>; 'Barone, Stan' <Barone.Stan@epa.gov>; 'Nguyen, Nhan' <Nguyen.Nhan@epa.gov>; 'Selby-Mohamadu, Yvette' <Selby-Mohamadu.Yvette@epa.gov>; 'Brinkerhoff, Chris' <Brinkerhoff.Chris@epa.gov>; 'Scheifele, Hans' <Scheifele.Hans@epa.gov>; 'Hanley, Mary' <Hanley.Mary@epa.gov>; 'Bertrand, Charlotte' <Bertrand.Charlotte@epa.gov>; 'Dourson, Michael' <dourson.michael@epa.gov>; 'Schlosser, Paul' <Schlosser.Paul@epa.gov>
Cc: 'Pierce, Alison' <Pierce.Alison@epa.gov>; 'Hasan, Jafrul' <Hasan.Jafrul@epa.gov>; 'Oxendine, Sharon' <Oxendine.Sharon@epa.gov>; 'Fehrenbacher, Cathy' <Fehrenbacher.Cathy@epa.gov>; 'Wolf, Joel' <Wolf.Joel@epa.gov>; 'Brown, Judith' <Brown.Judith@epa.gov>; 'Ortiz, Julia' <Ortiz.Julia@epa.gov>
Subject: RE: NMP Risk Evaluation (files part 2-Dupont 1990)

Please note that I am having problems with transmitting Dupont 1990. I will have to split the document into various pieces to send it by email. It is a huge file that can't be sent as is. I am troubleshooting and will try to send today or early Monday morning.

****

Iris A. Camacho, Ph.D.

Senior Science Advisor (on detail)

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Email: camacho.iris@epa.gov

From: Camacho, Iris
Sent: Friday, December 01, 2017 11:15 AM
To: Beck, Nancy <beck.nancy@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Mottley, Tanya <Mottley.Tanya@epa.gov>; Henry, Tala <Henry.Tala@epa.gov>; Barone, Stan <Barone.Stan@epa.gov>; Nguyen, Nhan <Nguyen.Nhan@epa.gov>; Selby-Mohamadu, Yvette <Selby-Mohamadu.Yvette@epa.gov>; Brinkerhoff, Chris <Brinkerhoff.Chris@epa.gov>; Scheifele, Hans <Scheifele.Hans@epa.gov>; Hanley, Mary <Hanley.Mary@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Schlosser, Paul <Schlosser.Paul@epa.gov>
Cc: Pierce, Alison <Pierce.Alison@epa.gov>; Hasan, Jafrul <Hasan.Jafrul@epa.gov>; Oxendine, Sharon <Oxendine.Sharon@epa.gov>; Fehrenbacher, Cathy <Fehrenbacher.Cathy@epa.gov>; Wolf, Joel <Wolf.Joel@epa.gov>; Brown, Judith <Brown.Judith@epa.gov>; Ortiz, Julia <Ortiz.Julia@epa.gov>
Subject: RE: NMP Risk Evaluation (files part 1)

Attached are the files that were requested yesterday at the briefing related to the studies and PBPK supplementary information informing the 2015 risk assessment.

Joel will have to send the remaining files related to the risk management activities.

I will send you another email (files part 2) submitting Dupont (1990). It is a large file that I can't attach to this email. Let me know if you need additional files.

****

Iris A. Camacho, Ph.D.

Senior Science Advisor (on detail)

Office of Pollution Prevention and Toxics

U.S. Environmental Protection Agency

William Jefferson Clinton Building East, 6308-A

Washington, DC 20460

Phone: 202 - 564 - 1229

Work hours: 8:00 am – 4:30 pm

Work cell phone: 202-304-8648

Telework phone: Ex. 6 - Personal Privacy

Email: camacho.iris@epa.gov

-----Original Appointment-----
From: Beck, Nancy
Sent: Tuesday, October 10, 2017 8:56 AM
To: Beck, Nancy; Morris, Jeff; Mottley, Tanya; Henry, Tala; Barone, Stan; Camacho, Iris; Nguyen, Nhan; Selby-Mohamadu, Yvette; Brinkerhoff, Chris; Scheifele, Hans; Hanley, Mary; Bertrand, Charlotte; Dourson, Michael; Schlosser, Paul
Cc: Pierce, Alison; Hasan, Jafrul; Oxendine, Sharon; Fehrenbacher, Cathy; Wolf, Joel; Brown, Judith; Ortiz, Julia
Subject: NMP Risk Evaluation
When: Thursday, November 30, 2017 1:00 PM-2:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where: DCRoomEast3156/DC-EPA-EAST-OCSPP (Call in number Ex. 6 - Personal Privacy ccess code Ex. 6 - Personal Privacy

POC for meeting materials: Hans Scheifele

To: Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]
From: Dourson, Michael
Sent: Wed 11/22/2017 4:58:33 PM
Subject: contact

Nancy and Charlotte

Here you go: Ex. 6 - Personal Privacy 2.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

To: Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]; Hanley, Mary[Hanley.Mary@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Wise, Louise[Wise.Louise@epa.gov]; Strong, Jamie[Strong.Jamie@epa.gov]; Flaherty, Colleen[Flaherty.Colleen@epa.gov]; Ohanian, Edward[Ohanian.Edward@epa.gov]
From: Dourson, Michael
Sent: Fri 12/1/2017 12:07:00 AM
Subject: Emmett et al. 2006
Emmett et al., 2006, Table 5 Results in part.xlsx

Dear Colleagues

Here is a quick pictorial look at some information from the Emmett et al. (2006) paper. Looking forward to our briefing tomorrow.

Cheers!

Michael…

… L. Dourson, PhD., DABT, FATS, FSRA

Senior Advisor to the Administrator

U.S. Environmental Protection Agency

dourson.michael@epa.gov

202-564-2463

www.epa.gov

From: Bertrand, Charlotte

Sent: Wednesday, November 29, 2017 2:06 PM
To: Hanley, Mary <Hanley.Mary@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>
Subject: Fwd: Slides for briefing on OW PFOA and PFOS HAs

Sent from my iPhone

Begin forwarded message:

From: "Strong, Jamie" <Strong.Jamie@epa.gov>
To: "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>
Cc: "Flaherty, Colleen" <Flaherty.Colleen@epa.gov>
Subject: RE: Slides for briefing on OW PFOA and PFOS HAs

Charlotte,

I apologize, but there is a comment bubble in the slide deck I sent you. Can you please switch out for this clean version.

Thanks,
Jamie

From: Bertrand, Charlotte
Sent: Tuesday, November 28, 2017 8:10 AM
To: Strong, Jamie <Strong.Jamie@epa.gov>
Cc: Hanley, Mary <Hanley.Mary@epa.gov>; Nagle, Deborah <Nagle.Deborah@epa.gov>; Burneson, Eric <Burneson.Eric@epa.gov>; Behl, Betsy <Behl.Betsy@epa.gov>; Flaherty, Colleen <Flaherty.Colleen@epa.gov>; Miller, Gregory <Miller.Gregory@epa.gov>; Donohue, Joyce <Donohue.Joyce@epa.gov>
Subject: RE: Slides for briefing on OW PFOA and PFOS HAs

Thank you, look forward to seeing you all on Friday.

Best,

Charlotte

From: Strong, Jamie
Sent: Tuesday, November 28, 2017 8:08 AM
To: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Cc: Hanley, Mary <Hanley.Mary@epa.gov>; Nagle, Deborah <Nagle.Deborah@epa.gov>; Burneson, Eric <Burneson.Eric@epa.gov>; Behl, Betsy <Behl.Betsy@epa.gov>; Flaherty, Colleen <Flaherty.Colleen@epa.gov>; Miller, Gregory <Miller.Gregory@epa.gov>; Donohue, Joyce <Donohue.Joyce@epa.gov>
Subject: Slides for briefing on OW PFOA and PFOS HAs

Charlotte,

Please find attached OW's slides for the briefing on the health advisories for PFOA and PFOS scheduled for Friday. Please let me know if you need anything further.

Thank you,

Jamie Strong

Chief Human Health Risk Assessment Branch

Health and Ecological Criteria Division, 4304-T

Office of Science and Technology, Office of Water

United States Environmental Protection Agency

1200 Pennsylvania Avenue, NW

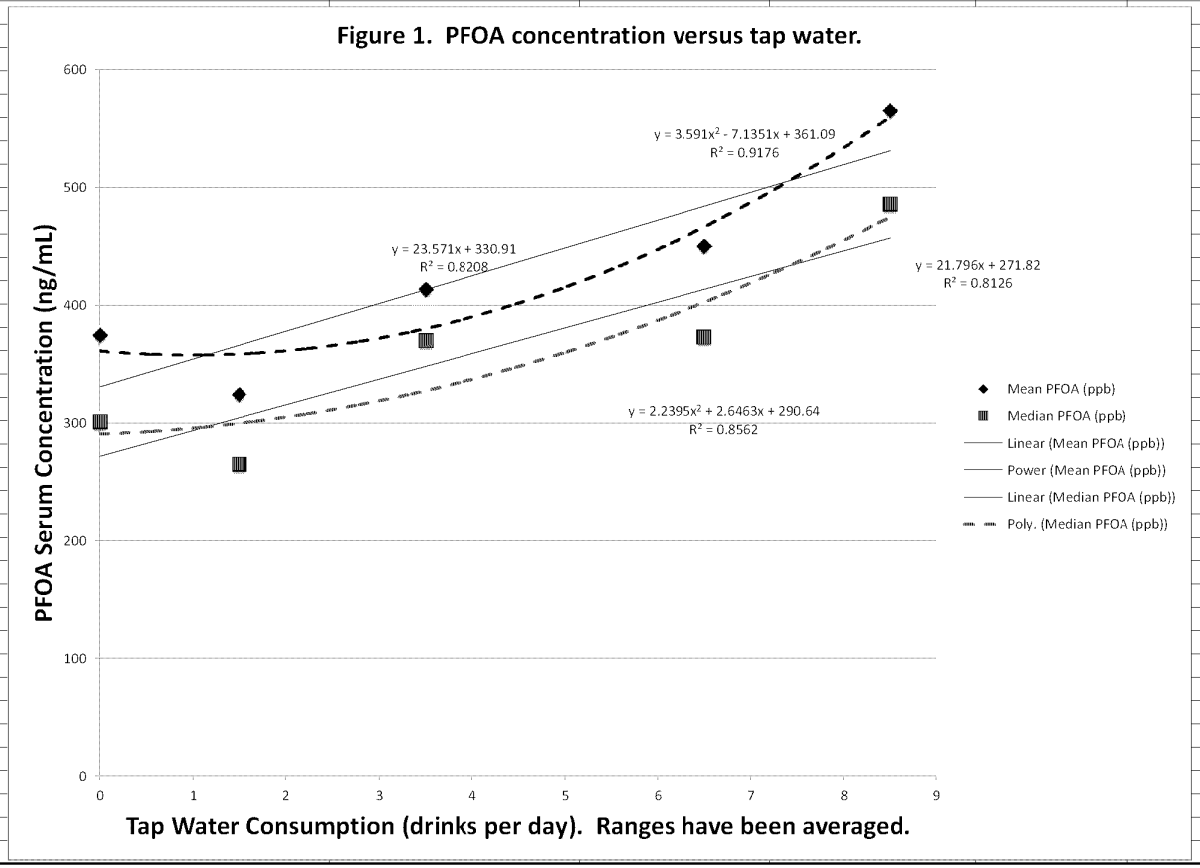
Washington DC 20460

phone: 202.566.0056

fax: 202.566.1140

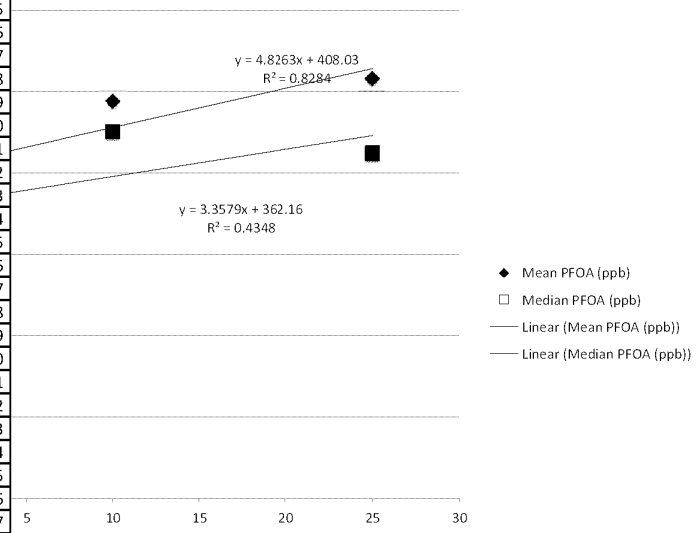
	A	B	C	D	E	F	G
1	Emmett et al. (2006) Table 5, page 23.						
2							
3		Tap Water Drinks per Day	Mean PFOA (ppb)	Median PFOA (ppb)			
4		0	374	301			
5		1.5	324	265			
6		3.5	413	370			
7		6.5	450	373			
8		8.5	565	486			
9							
10							

Figure 1. PFOA concentration versus tap water.



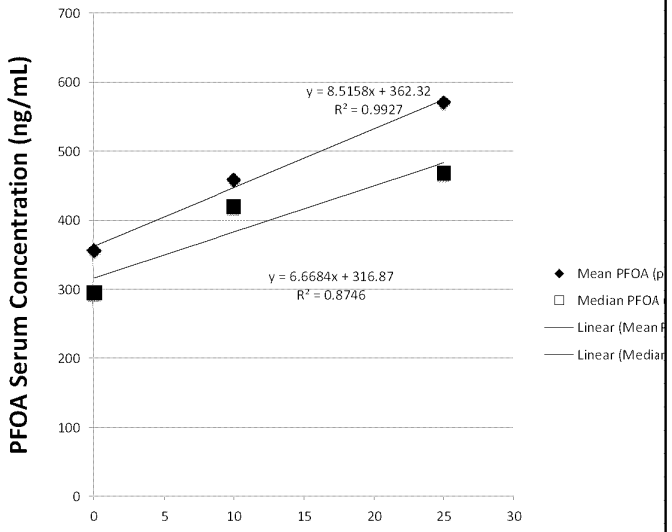
	H	I	J	K	L	M	N	O
1								
2								
3	Local Meat Servings/week	Mean PFOA (ppb)	Median PFOA (ppb)			Local Vegetables Servings/week	Mean PFOA (ppb)	Median PFOA (ppb)
4	0	389	329			0	356	295
5	10	488	451			10	458	420
6	25	516	424			25	571	469
7								
8								
9								
10								
11								

igure 2. PFOA concentration versus local meat.



Meat Servings (meals per week). Ranges have been averaged.

Figure 3. PFOA concentration versus local vegetables



Local Vegetable Servings (meals per week). Ranges have been averaged.

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ED_001803B_00003840_00000002

To: Hanley, Mary[Hanley.Mary@epa.gov]; Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]
From: Dourson, Michael
Sent: Thur 11/30/2017 11:49:40 PM
Subject: RE: Use this Version: Draft Response to SEPW Minority Letter and QFRs
MDmdFINAL Dourson QFRs 11.14.2017.MH.CB.docx (003).docx
mdDourson Response to SEPW Minority Letter.draft 11.05.17.MCH.docx

Mary

Thanks for keeping this on the burner. I have tweaked the version trying to mollify several of the statements. See what you all think.

Michael

From: Hanley, Mary
Sent: Thursday, November 30, 2017 10:53 AM
To: Beck, Nancy <Beck.Nancy@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>
Subject: FW: Use this Version: Draft Response to SEPW Minority Letter and QFRs
Importance: High

Hello, the latest version of the letter is attached. Mike there are some comments for your attention. I also added the Bodine letter v Ex. 5 - Deliberative Process Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Cheers

M

From: Hanley, Mary
Sent: Wednesday, November 29, 2017 5:29 PM
To: Beck, Nancy <beck.nancy@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Michael Dourson (dourson.michael@epa.gov) <dourson.michael@epa.gov>

Subject: Use this Version: Draft Response to SEPW Minority Letter and QFRs
Importance: High

Nancy, Charlotte, Mike:

Please use the QFR version attached for further editing. It is substantively the same as the last version Nancy has (with Charlotte's comments) but I have corrected some formatting problems.

Also, please review and comment on the response to the letter attached. I don't think I received edits on it yet.

In addition, I have included Mike's initial draft responses to the incoming letter. Note that since that time Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process Let me know of any questions.

Cheers

M

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 4:45:56 PM
Subject: RE: Neonicotinoid Briefing

Thanks!

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 12:17 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Subject: RE: Neonicotinoid Briefing

Louise can attend the 1pm meeting for us. Ex. 5 - Deliberative Process
Ex. 5 - Deliberative Process
You should get an invited to standing 8am meetings for political leadership meetings with the Administrator. If you don't have those yet, ask Hayley Ford to add you to the list.

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
beck.nancy@epa.gov

-----Original Appointment-----
From: Dourson, Michael
Sent: Wednesday, October 18, 2017 12:13 PM
To: Bertrand, Charlotte; Beck, Nancy
Subject: Tentative: Neonicotinoid Briefing
When: Monday, October 23, 2017 1:00 PM-2:00 PM (UTC-05:00) Eastern Time (US & Canada).
Where: PY-12100

Charlotte, I also have a senior staff briefing with Mr. Pruitt.

Nancy, which meeting get preference? I assume you can handle Administrator Pruitt's meeting.

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 11/22/2017 2:45:14 PM
Subject: RE: ATSDR

Thanks.

From: Beck, Nancy
Sent: Wednesday, November 22, 2017 9:24 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: ATSDR

We can talk about it

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

On Nov 22, 2017, at 9:21 AM, Dourson, Michael <dourson.michael@epa.gov> wrote:

Nancy

What is this about please? Thanks!

Mike

From: Beck, Nancy
Sent: Wednesday, November 22, 2017 8:40 AM
To: Vogel, Dana <Vogel.Dana@epa.gov>; Keigwin, Richard <Keigwin.Richard@epa.gov>
Cc: Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; Wise, Louise <Wise.Louise@epa.gov>; Dourson, Michael <dourson.michael@epa.gov>; Keller, Kaitlin <keller.kaitlin@epa.gov>

Subject: ATSDR

I had a nice chat with Pete Breysse this morning. He was surprised to hear that their draft 800 page tox profile makes a finding of not enough information. I think he was also

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process

Ex. 5 - Deliberative Process I expect as a next step John Decker will reach out to Dana next week.

Lets chat after the holiday. I hope I didn't get anything too wrong.

Nancy

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 4:31:41 PM
Subject: RE: K-Day Program is attached. Arnold

Thanks!

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 12:24 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: K-Day Program is attached. Arnold

There is a shuttle that runs there, however, we have access to a car (you and I) and we can use that. I think I have it scheduled for around 11:50 as the real festivities once start til noon.

Once the car is set I will forward the invite to you.

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 12:22 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: K-Day Program is attached. Arnold

Nancy

I have ethics training with Justina from 10 to 11. Afterwards I will go over to PY. So what is the best way?

Mike

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 10:53 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Fwd: K-Day Program is attached. Arnold

Nancy B. Beck, Ph.D., DABT
Deputy Assistant Administrator, OCSPP
P: 202-564-1273
M: Ex. 6 - Personal Privacy
Beck.Nancy@epa.gov

Begin forwarded message:

From: "Layne, Arnold" <Layne.Arnold@epa.gov>
To: "OPP Division Directors" <OPP_Division_Directors@epa.gov>, "OPP Associate and Deputy Directors" <OPP_Associate_and_Deputy_Directors@epa.gov>, "Beck, Nancy" <Beck.Nancy@epa.gov>, "Hughes, Hayley" <hayley.hughes@hq.dhs.gov>, "Barone, Stan" <Barone.Stan@epa.gov>, "Graves, Inza" <Graves.Inza@epa.gov>, "Morris, Jeff" <Morris.Jeff@epa.gov>, "Hartman, Mark" <Hartman.Mark@epa.gov>, "Mottley, Tanya" <Mottley.Tanya@epa.gov>, "Simon, Nigel" <Simon.Nigel@epa.gov>, "Breen, Barry" <Breen.Barry@epa.gov>, "Woolford, James" <Woolford.James@epa.gov>, "Ross, Mary" <Ross.Mary@epa.gov>, "Morales, Oscar" <Morales.Oscar@epa.gov>, "Bertrand, Charlotte" <Bertrand.Charlotte@epa.gov>, "OPPT Division Directors Deputy Directors" <OPPT_Division_Directors_Deputy_Directors@epa.gov>, "OPPT Division Directors" <OPPT_Division_Directors@epa.gov>, "Johnson, Barnes" <Johnson.Barnes@epa.gov>
Cc: "Stewart, Troy" <Stewart.Troy@epa.gov>
Subject: K-Day Program is attached. Arnold

Wear PINK! And practice the dance!! Given the crowd size, we will take the PINK photo outside at noon then reenter the building for the managers' dance.

Arnold E. Layne

Deputy Director for Management

EPA Chief Customer Experience Officer for OPP

EPA OCSPP Lead for Zika

Office of Pesticide Programs

US Environmental Protection Agency

703-347-8222

"Nobody cares how much you know, until they know you care about them!" Zig Ziglar

From: Layne, Arnold
Sent: Monday, October 16, 2017 1:26 PM
To: OPP Division Directors <OPP_Division_Directors@epa.gov>; OPP Associate and Deputy Directors <OPP_Associate_and_Deputy_Directors@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Hughes, Hayley <hayley.hughes@hq.dhs.gov>; Barone, Stan <Barone.Stan@epa.gov>; Graves, Inza <Graves.Inza@epa.gov>; Morris, Jeff <Morris.Jeff@epa.gov>; Hartman, Mark <Hartman.Mark@epa.gov>; Mottley, Tanya <Mottley.Tanya@epa.gov>; Simon, Nigel <Simon.Nigel@epa.gov>; Breen, Barry <Breen.Barry@epa.gov>; Woolford, James <Woolford.James@epa.gov>; Ross, Mary <Ross.Mary@epa.gov>; Morales, Oscar <Morales.Oscar@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>; OPPT Division Directors Deputy Directors <OPPT_Division_Directors_Deputy_Directors@epa.gov>; OPPT Division Directors <OPPT_Division_Directors@epa.gov>; Johnson, Barnes <Johnson.Barnes@epa.gov>
Cc: Marshall, Kim <Marshall.Kim@epa.gov>; Layne, Arnold <Layne.Arnold@epa.gov>; Stewart, Troy <Stewart.Troy@epa.gov>
Subject: ACTION - K-Day Practice Video!
Importance: High

Dear Colleagues,

Below is the youtube link to the Macarena with music from which to practice for the K-day managers' performance. See you Thursday, October 19, 2017, promptly at 11 AM at Potomac Yard South in the first floor conference room.

Remember to wear PINK in honor of Breast Cancer Awareness month. We will take our annual Wear Pink photo at noon immediately followed by the IO Senior leadership, OCSPP/OLEM/ORD SESers, and their Deputies/Associates Macarena dance performance. You will be directed where to go to take the PINK photo.

Please don't tell the staff what dance we are doing. Let's keep that a secret.

We can do this team and look good doing it!! See you Thursday in PINK. All my best, Arnold

https://www.youtube.com/watch?v=i1im74-XgYA

Arnold E. Layne

Deputy Director for Management

EPA Chief Customer Experience Officer for OPP

EPA OCSPP Lead for Zika

Office of Pesticide Programs

US Environmental Protection Agency

703-347-8222

"Nobody cares how much you know, until they know you care about them!" Zig Ziglar

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 4:30:41 PM
Subject: RE: Potomac Yards Shuttle

Thanks!

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 12:26 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Potomac Yards Shuttle

https://www.epa.gov/sites/production/files/2016-04/documents/shuttlebus.pdf

****

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator

Office of Chemical Safety and Pollution Prevention

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

To: Munoz, Charles[munoz.charles@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Fugh, Justina[Fugh.Justina@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 12:06:00 PM
Subject: RE: Introductory briefing

Thanks!

From: Munoz, Charles
Sent: Wednesday, October 18, 2017 8:04 AM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>; Fugh, Justina <Fugh.Justina@epa.gov>
Subject: RE: Introductory briefing

It is.

Charles Munoz

White House Liaison

Environmental Protection Agency

202-380-7967

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 8:03 AM
To: Munoz, Charles <munoz.charles@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>; Fugh, Justina <Fugh.Justina@epa.gov>
Subject: RE: Introductory briefing

Charles

I have an ethics briefing tomorrow on my calendar at 10 am with your name on it. Is this with Justina Fugh?

Cheers!

Michael

From: Fugh, Justina
Sent: Tuesday, October 17, 2017 9:48 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Well, gosh, that is me, but it's not on my calendar! I'll add it myself … Thursday, October 19 at 10 am, correct?

From: Dourson, Michael
Sent: Tuesday, October 17, 2017 9:26 PM
To: Fugh, Justina <Fugh.Justina@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Justina

I have an ethics briefing slated for this time, in room 4308. I presumed this was you. Please confirm at your leisure.

Cheers!

Michael

If you cannot explain it simply, you do not understand it well enough---Albert Einstein

From: Fugh, Justina
Sent: Tuesday, October 17, 2017 6:23 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Howdy! We should find one hour so that I can give you your new employee ethics briefing! Are you still free at 10 on Thursday? If not, then we can choose some time Friday afternoon, okay?

Justina Fugh | Senior Counsel for Ethics | Office of General Counsel | US EPA | Mail Code 2311A | Room 4308 North, William Jefferson Clinton Federal Building | Washington, DC 20460 (for ground deliveries, use 20004 for the zip code) | phone 202-564-1786 | fax 202-564-1772

From: Dourson, Michael
Sent: Tuesday, October 17, 2017 9:56 AM
To: Fugh, Justina <Fugh.Justina@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: Introductory briefing

Justina

I will be in the EPA HQ building tomorrow and for the rest of the week. When would be a good time to get together? My schedule tomorrow is open from about 1:30 to 4 pm. Thusday, I am open at 10 am and after 4 pm. Friday, I am open after 9:30 and for the whole day.

Cheers!

Michael

To: Munoz, Charles[munoz.charles@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Fugh, Justina[Fugh.Justina@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 12:03:04 PM
Subject: RE: Introductory briefing

Charles

I have an ethics briefing tomorrow on my calendar at 10 am with your name on it. Is this with Justina Fugh?

Cheers!

Michael

From: Fugh, Justina
Sent: Tuesday, October 17, 2017 9:48 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Well, gosh, that is me, but it's not on my calendar! I'll add it myself … Thursday, October 19 at 10 am, correct?

From: Dourson, Michael
Sent: Tuesday, October 17, 2017 9:26 PM
To: Fugh, Justina <Fugh.Justina@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Justina

I have an ethics briefing slated for this time, in room 4308. I presumed this was you. Please confirm at your leisure.

Cheers!

Michael

If you cannot explain it simply, you do not understand it well enough---Albert Einstein

From: Fugh, Justina
Sent: Tuesday, October 17, 2017 6:23 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Howdy! We should find one hour so that I can give you your new employee ethics briefing! Are you still free at 10 on Thursday? If not, then we can choose some time Friday afternoon, okay?

Justina Fugh | Senior Counsel for Ethics | Office of General Counsel | US EPA | Mail Code 2311A | Room 4308 North, William Jefferson Clinton Federal Building | Washington, DC 20460 (for ground deliveries, use 20004 for the zip code) | phone 202-564-1786 | fax 202-564-1772

From: Dourson, Michael
Sent: Tuesday, October 17, 2017 9:56 AM
To: Fugh, Justina <Fugh.Justina@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: Introductory briefing

Justina

I will be in the EPA HQ building tomorrow and for the rest of the week. When would be a good time to get together? My schedule tomorrow is open from about 1:30 to 4 pm. Thusday, I am open at 10 am and after 4 pm. Friday, I am open after 9:30 and for the whole day.

Cheers!

Michael

Appendix A **Please read Privacy Act Statement and instructions on reverse before completing this form.**

United States Environmental Protection Agency
Washington, DC 20460

EPA TSCA CBI Access Request, Agreement, and Approval

Section I. – Access Request

1. Name (Last, First, MI)	2. 9-Digit UID Number (e.g., SSN)	3. Telephone Number
Dourson, Michael, L	Ex. 6 - Personal Privacy	(202) 564-2463
4. Requestor (Agency/Office/Division/Branch)	5. Document Control Officer (DCO)	6. DCO Telephone Number

7. TSCA Sections for which access is required. Check all that apply. Use blank space to request other sections not listed.
ALL ☐ -OR- 4 ☐ 5 ☐ 6 ☐ 8 ☐ 12 ☐ 13 ☐ 21 ☐ _____

8. Justification for TSCA CBI access. Select appropriate code from instructions on reverse side. (Check one for all that apply).
A ☐ B ☐ C ☐ D ☐ Other ☐ List Justification on reverse side

Section II. – Contract Information - Contractor Employees Only

9. Employer's Name	10a. Employer's Address	10b. City	10c. ST	10d. Zipcode
11. Contract Number	12. EPA Project Officer	13. EPA Project Officer Telephone		

Section III. – OPPT Secure Storage Area Access – HQ Federal and HQ Contractor Employees Only

14. Check if EPA ID Badge. Badge is required.
☐ Yes (New) ☐ Need Replacement ☐ No (List Present EPA ID Badge Number _____)

15. List OPPT Restricted areas by Division to which physical access is required.

Home Division (24 hour access)	Other Divisions (6A.M. – 6P.M. only)	Access to CBIC Only	IMD (DCO and IMD Computer Rms.)

16. List OPPT areas by Division and Room Number for which Alarm Activation/Deactivation Authority is requested.

Section IV. – Confidentiality Agreement

I understand that I will have access to certain Confidential Business Information submitted under the Toxic Substances Control Act (TSCA, 15 USC 2601 et seq.). This access has been granted in accordance with my official duties relating to Environmental Protection Agency programs.

I understand that TSCA CBI may be used only in connection with my official duties and may not be disclosed except as authorized by TSCA and Agency regulations. I have received a copy of, and understand the procedures set forth in, the TSCA CBI Protection Manual. I agree that I will treat any TSCA CBI furnished to me as confidential and that I will follow these procedures.

I understand that under section 14(d) of TSCA (15 USC 2513(d)), I am liable for a possible fine of up to $5,000 and/or imprisonment for up to one year if I willfully disclose TSCA CBI to any person not authorized to receive it. In addition, I understand that I may be subject to disciplinary action for violation of this agreement with penalties ranging up to and including dismissal.

I understand that my obligation to protect TSCA CBI, which has been disclosed to me as part of my official job duties, continues after either termination of my assignment or termination of my employment.

I certify that the statements I have made on this form and all attachments thereto are true, accurate, and complete. I acknowledge that any knowingly false or misleading statement may be punishable by fine or imprisonment or both under applicable law.

17. Signature of Employee	18. Date
Michael L. Dourson	10/18/2017

Section V. – Requesting Official Approval

19. TSCA CBI Security Briefing Date	20. Name and Signature of Requesting Official. (Immediate Supervisor – EPA Project Officer for Contractors) As the immediate supervisor of (or the EPA Project Officer for) the above mentioned employee, I certify he/she has successfully completed a TSCA CBI Security Briefing on the date shown.	
	Name / Signature	21. Date

22. Date Received	23. Approved (TSCA Security Official Signature)	24. Approval Date

DCO Code	Barcode	Status Code	Alarm Zones	Data Entry Date and Initials
				1. 2.

EPA Form 7740-6 (Rev. 10-03). Replaces previous version of 7740-6 and 7740-6A.

Paperwork Reduction Act Notice

The public reporting burden for the collection of information is estimated to average .84 hours per response. This estimate includes time for reviewing instructions, gathering and maintaining the needed data, and completing and reviewing the collection of information. Send comments regarding the burden estimate or any other aspect of this collection of information to the Director, Collection Strategies Division, US Environmental Protection Agency (2822T), 1200 Pennsylvania Ave., NW, Washington DC 20460, and to the Office of Information and Regulatory Affairs, Office of Management and Budget, Washington, DC 20503, marked ATTENTION: Desk Officer for EPA. Include the OMB No. identified on page 1 in any correspondence. Do not send the completed form to this address. Submit the form in accordance with the instructions in the CBI Manual.

Privacy Act Statement

Furnishing your Social Security Number is voluntary, but encouraged. The information on this form is used by EPA to maintain a record of those persons cleared for access to TSCA Confidential Business Information (CBI) and to maintain the security of TSCA CBI.

Disclosure of information from this form may be made to the Office of Pollution Prevention and Toxics (OPPT) contractors in order to carry out functions for EPA compatible with the purpose for which this information is collected; to other Federal agencies when they possess TSCA CBI and need to verify clearance to EPA and EPA contractor employees for access; to the Department of Justice when related to litigation or anticipated litigation involving the records or the subject matter of the records; to the appropriate Federal, State or local agency charged with enforcing a statute or regulation, violation of which is indicated by a record in this system; where necessary, to a State, Federal or local agency maintaining information pertinent to hiring, retention, or clearance of an employee, letting of a contract, or issuance of a grant or other magistrate or administrative tribunal; in the course of litigation under TSCA; and to a member of Congress acting on behalf of an individual to whom records in this system pertain.

Instructions for Form Completion

Section I – To be completed by all

1. List Full Name
2. List 9-Digit ID (e.g., SSN)
3. List Telephone number of person in item 1
4. List Full Acronym of Requesting Office (i.e. EPA Office in which the individual works or for contractor employees, the EPA Office with whom the contract is with)
5. List the immediate Document Control Officer for the office in which the individual works
6. List the telephone number of the Document Control Officer
7. Check the TSCA Sections for which access is requested or check ALL if applicable
8. Circle the appropriate Access Justification Code

A *Employee is an EPA employee or EPA contractor employee whose work assignments involve the New and/or Existing Chemical Programs of TSCA. Hence access to the TSCA sections listed in item 7 of this form is required in performance of his/her duties*.

B. *Employee is an EPA employee or EPA contractor employee whose work entails the administration of computer systems housing TSCA CBI. Hence access to the TSCA sections listed in item 7 of this form is required.*

C. *Employee is an EPA employee or EPA contractor employee whose work entails physical security or maintenance for TSCA CBI secure storage areas. Although employee will not actually work with any TSCA CBI materials, access to the TSCA sections listed in item 7 of this form is required.*

D. List Justification here_____

_____

_____

_____

_____

_____

_____

Section II – To be completed by Contractor Employees only

9. List Employer's name
10a-d. List Employer's address
11. List Contract number
12. List EPA Project Officer's name
13. List EPA Project Officer's telephone number

Section III – To be completed by HQ Federal and HQ Contractor employees only

NOTE: *These procedures apply only to employees requiring access to OPPT Secure Storage areas. All others follow standard Agency procedures.*

14. Check either box a, b, c or (c&d) for EPA ID badge or Contractor Building Pass. If box c is checked, write in badge number.

a. Yes - Check if new employee getting first EPA ID Badge. (New programmed badge and barcode)

b. Need Replacement - Check if replacement ID Badge is needed (replacement badge and barcode)

c. No - Existing badge needs programming. List ID Badge no.

15. Check and list OPPT secured areas for which access (via "RUSCO" electronic door control system) is required. List Division acronyms for the requested areas.

Home Division - List Division in which employee works

Other Divisions - List other OPPT Divisions for which unrestricted daytime access is requested

CBIC Only - To be checked for those who only need to access the Confidential Business Information Center.

IMD Areas - Employees who need to regularly access the IMD Document Control Office Suite should circle **DC0** in the fourth block. Only IMD staff and contractors who work in IMD computer rooms should circle **IMD Computer Rooms**.

16. List OPPT areas by Division and Room numbers for which Alarm Activation/Deactivation authority is requested. Generally, this is employees home Division only.

Section IV – To be completed by all

17. Employee Signature (must be original)
18. Signature Date

Section V – To be completed by all

19. Enter date employee attended TSCA CBI Security Briefing
20. Immediate Supervisor/EPA Project Officers name and sign.
21. Date of signature

Section VI – To be completed by OPPT Security

To: Hanley, Mary[Hanley.Mary@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]; Bertrand, Charlotte[Bertrand.Charlotte@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 10:43:24 AM
Subject: RE: Copy of TCE Work Plan Risk Assessment

Mary

Would you please be so kind and send me the external peer review report. The link in the document you sent for this report was not working.

Thanks!

Michael

If you cannot explain it simply, you do not understand it well enough---Albert Einstein

From: Hanley, Mary
Sent: Wednesday, October 18, 2017 5:19 PM
To: Dourson, Michael <dourson.michael@epa.gov>; Beck, Nancy <Beck.Nancy@epa.gov>; Bertrand, Charlotte <Bertrand.Charlotte@epa.gov>
Subject: Copy of TCE Work Plan Risk Assessment

Per your request.

Cheers
M

To: Fugh, Justina[Fugh.Justina@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 1:26:27 AM
Subject: RE: Introductory briefing

Justina

I have an ethics briefing slated for this time, in room 4308. I presumed this was you. Please confirm at your leisure.

Cheers!

Michael

If you cannot explain it simply, you do not understand it well enough---Albert Einstein

From: Fugh, Justina
Sent: Tuesday, October 17, 2017 6:23 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Introductory briefing

Howdy! We should find one hour so that I can give you your new employee ethics briefing! Are you still free at 10 on Thursday? If not, then we can choose some time Friday afternoon, okay?

Justina Fugh | Senior Counsel for Ethics | Office of General Counsel | US EPA | Mail Code 2311A | Room 4308 North, William Jefferson Clinton Federal Building | Washington, DC 20460 (for ground deliveries, use 20004 for the zip code) | phone 202-564-1786 | fax 202-564-1772

From: Dourson, Michael
Sent: Tuesday, October 17, 2017 9:56 AM
To: Fugh, Justina <Fugh.Justina@epa.gov>
Cc: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: Introductory briefing

Justina

I will be in the EPA HQ building tomorrow and for the rest of the week. When would be a good time to get together? My schedule tomorrow is open from about 1:30 to 4 pm. Thusday, I am open at 10 am and after 4 pm. Friday, I am open after 9:30 and for the whole day.

Cheers!

Michael

To: Bowman, Liz[Bowman.Liz@epa.gov]
From: Dourson, Michael
Sent: Thur 10/19/2017 1:56:17 AM
Subject: RE: Hello

Thanks!

From: Bowman, Liz
Sent: Wednesday, October 18, 2017 3:22 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: Hello

Yes we did

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 12:16 PM
To: Bowman, Liz <Bowman.Liz@epa.gov>
Subject: RE: Hello

Liz

Thanks! I presume that you also responded to the reporter looking for the sick-child, mother, dead dog story that I described briefly at my hearing?

Cheers!

Michael

From: Bowman, Liz
Sent: Wednesday, October 18, 2017 11:50 AM

To: Dourson, Michael <dourson.michael@epa.gov>
Subject: Re: Hello

We responded to him w a list of others who also started pre vote.

Sent from my iPhone

On Oct 18, 2017, at 11:47 AM, Dourson, Michael <dourson.michael@epa.gov> wrote:

> Liz
>
> Please let me know if you need any help. Cheers!
>
> Michael
>
> **From:** Alex Guillen [mailto:aguillen@politico.com]
> **Sent:** Wednesday, October 18, 2017 8:45 AM
> **To:** Dourson, Michael <dourson.michael@epa.gov>
> **Subject:** Hello
>
> Hi Michael,
>
> My name is Alex Guillén, and I'm an energy reporter with Politico. I saw a report that you've already started at EPA and wanted to confirm with you that you're a special adviser on chemical and pesticide issues. Are you detailed to OCSPP, or are you working out of the administrator's office for now? What will you work on until you are confirmed?
>
> Thank you,
>
> Alex Guillén

Energy Reporter

POLITICO ***Pro***

(o) 703.341.4619 | (c) 571.839.6243

aguillen@politico.com | @alexcguillen

To: Frye, Tony (Robert)[frye.robert@epa.gov]
From: Dourson, Michael
Sent: Tue 10/17/2017 6:14:43 PM
Subject: RE: Sullivan Bio

Tony

Thanks got it.

Michael

From: Frye, Tony (Robert)
Sent: Tuesday, October 17, 2017 1:34 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Cc: Shimmin, Kaitlyn <shimmin.kaitlyn@epa.gov>; Palich, Christian <palich.christian@epa.gov>
Subject: Sullivan Bio

Hello Dr. Dourson,

Attached, please find a meeting memo in advance of your telephone call with Senator Sullivan. Feel free to let us know if you have any other thoughts or questions.

Best,

Tony

Tony Frye

Special Assistant to the Deputy Associate Administrator

Office of Congressional & Intergovernmental Affairs

Environmental Protection Agency

Phone: 202.564.0640

Cell: Ex. 6 - Personal Privacy

To: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Wed 10/18/2017 5:21:26 PM
Subject: RE: Allowed: Sharing request: Calendar

Yes, this system is ancient.

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 1:13 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: Allowed: Sharing request: Calendar

Ahhhh. Were you a Mac person?

Its pretty easy—you will catch on and the premier support group are rockstars!

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 1:11 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Allowed: Sharing request: Calendar

Got it. Thanks. Part of the problem is my computer is a Dell. Not at all use to this.

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 1:02 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: Allowed: Sharing request: Calendar

Hmm. I wont let me resend, but if you bring your computer with you to a meeting (and your ID) I can show you how to change permissions.

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 12:43 PM
To: Beck, Nancy <Beck.Nancy@epa.gov>
Subject: RE: Allowed: Sharing request: Calendar

Nancy

Sure, please send me the request again. I could not find your prior email. And no, no one is helping me with schedules, but perhaps because I have not asked.

Mike

From: Beck, Nancy
Sent: Wednesday, October 18, 2017 12:28 PM
To: Dourson, Michael <dourson.michael@epa.gov>
Subject: RE: Allowed: Sharing request: Calendar

Mike,

Can you give me full access to your calendar so I can see appointments that I will need to schedule around? I think the option is "full details".

Right now I can just see free/busy but not the event.

Also, is there a scheduler that is helping you out?

Thanks.

Nancy

Nancy B. Beck, Ph.D., DABT

Deputy Assistant Administrator, OCSPP

P: 202-564-1273

M: Ex. 6 - Personal Privacy

beck.nancy@epa.gov

From: Dourson, Michael
Sent: Wednesday, October 18, 2017 11:43 AM
To: Beck, Nancy
Subject: Allowed: Sharing request: Calendar

Microsoft Exchange Calendar: Dourson, Michael - Calendar

Dourson, Michael (dourson.michael@epa.gov) has allowed you to view his or her Calendar.

To: Fugh, Justina[Fugh.Justina@epa.gov]
Cc: Beck, Nancy[beck.nancy@epa.gov]
From: Dourson, Michael
Sent: Tue 10/17/2017 1:56:18 PM
Subject: Introductory briefing

Justina

I will be in the EPA HQ building tomorrow and for the rest of the week. When would be a good time to get together? My schedule tomorrow is open from about 1:30 to 4 pm. Thusday, I am open at 10 am and after 4 pm. Friday, I am open after 9:30 and for the whole day.

Cheers!

Michael